South African Medical Journal : Suid-Afrikaanse Tydskrif vir Geneeskunde

Cape Town, 26 August 1961 Volume 35 No. 34 Deel 35 Kaapstad, 26 Augustus 1961

SEX CHROMOSOMES AND ABNORMAL SEX DEVELOPMENT*

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In the last decade interest has been revived in the chromosomal constitution of man, following the demonstration of its relation to various clinical syndromes, congenital abnormalities and malignancy. The chromosome number, which had been accepted as 48, was shown in 1956 by workers[1a,b] from different laboratories to be 46, i.e. 22 pairs of autosomes plus 2 sex chromosomes.

These recent advances have resulted from the recognition of the sex chromatin body, the application of tissue-culture methods, and improvements in the technique of making squash preparations. This field is still in its infancy and the evaluation and interpretation of the findings at this stage is somewhat involved and difficult, with many apparent contradictions.

SEXUAL DEVELOPMENT

This depends on the initial processes of determination and subsequent differentiation.

Sex Determination

Sexual development in the embryo is primarily dependent on the chromosomal constitution of the zygote, i.e. it is genetically determined by the sex chromosomes and probably by specific autosomes on which the genes which transmit the sex characters are located ('primary inductors').

Sex Differentiation

The zygotic sex dictates organ development along either the male or the female pathway.

1. *Foetal Differentiation—Primary Sex Development*

(*a*) *Gonad.* The differentiation of the gonad is determined by the zygotic sex of the embryo. During embryogenesis the germ cells migrate from the yolk sac to the undifferentiated gonad. Failure to do so may be a factor in abnormal sexual development. In man it is possible that the testis produces morphogenetic substances ('secondary inductors') which cause male differentiation, and that female differentiation results from the absence of these inductors and not from the presence of ovarian substances. Jost[2] showed this in the embryos of lower animals in castration experiments.

(*b*) *Internal accessory sex organs.* In the male these are differentiated from the Wolffian system and in the female from the Mullerian system.

(*c*) *External genitalia.* The differentiation of the external genitalia is probably hormone dependent, the hormones being the androgens produced by the foetal testis and adrenal. It appears to be independent of internal differentiation.

2. *Pubertal Differentiation—Secondary Sex Development*

This is hormone dependent ('tertiary inductors'), the source of the hormones being both gonadal and extragenital endocrine glands. Both the male and female hormones are important.

Sex abnormalities can occur at any of the above stages of sexual development.

THE SEX OF THE INDIVIDUAL

This can be assessed at different levels.

Morphological Level

This includes the following:

1. *Chromosomal sex* at the nuclear level. Chromatin positive implies the presence of the sex chromatin body in a high proportion of nuclei, as in normal females. If it is absent the individual is chromatin negative, as in normal males.

2. *Gonadal sex.* This includes: (*a*) ovaries, (*b*) testes, (*c*) mixed—true hermaphrodite, and (*d*) neuter—undifferentiated gonad.

3. *Internal accessory sex organs.*

4. *External sex organs.*

In (3) and (4) these may be normal male or female, or may be mixed or ambiguous as in the true hermaphrodite or pseudohermaphrodite.

Physiological Level

The adult endocrine pattern responsible for the development and maintenance of secondary sex characteristics.

Psychological Level

1. Assigned sex at birth.
2. Sex of rearing, which is determined by assigned sex.
3. Psychological influences of the environment.

Synchronization

When all the above components are compatible and synchronize, the sexual development is normal male or female. If any single one is conflicting, the result is abnormal, and different clinical syndromes may present.

This discussion will be chiefly at the cellular level, i.e. the karyotype and chromosome number associated with recognized clinical abnormalities.

KARYOTYPES

Sex Chromatin

Barr and his co-workers in 1949[3] identified this structure in the nuclei of female inter-phase somatic cells as a more densely staining chromatin body approximately 1μ in diameter. It is most easily recognized when closely applied to

* Abridged from a lecture delivered at a Scientific Meeting of The South African Institute for Medical Research on 31 October 1960.

TABLE I. RECENT INVESTIGATIONS IN PATIENTS WITH CONFLICTING SEX COMPONENTS

Clinical syndrome	*Phenotype*	*Gonad*	*Internal genital organs*	*External genitalia*	*Secondary sex characteristics*	*Congenital anomalies*	*Nuclear sex*	*Chromosome number*	*Karyotype*
Klinefelter's syndrome[4,5]	Male—sterile	Testis, hypospermatogenesis and hyalinization of tubules	Male	Male	(1) Gynaecomastia + or −. (2) Absent or deficient facial hair	—	Chromatin $+_{ve}$	47	XXY
Klinefelter double male[6]							Chromatin $+_{ve}$	48	XXYY
Klinefelter mosaic[7,8]						Psychopath	Chromatin $+_{ve}$, some cells 2 sex chromatin bodies	47/46	XXY/XX mosaic
Klinefelter variant[9]						Primary amentia	Chromatin $+_{ve}$. Double chromatin bodies	48	XXXY
Turner's syndrome[10,11]	Female—usually sterile	(*a*) Rudimentary undifferentiated gonad, (*b*) ovarian stroma, + or − mesonephric ducts	Female—infantile	Female	Sexual infantilism	++	Chromatin $-_{ve}$	45	XO
Pure gonadal agenesis[12]		? Primitive gonadal ridges			Sexual infantilism	—	Chromatin $-_{ve}$	46	XY
Turner mosaic[13]							Chromatin $-_{ve}$ or weakly $+_{ve}$	45/46	XO/XX mosaic
Triplo-X female 'super-female'[14,15]	Female—(*a*) sterile, (*b*) fertile	Ovary	Female—(*a*) normal, (*b*) underdeveloped	Female—normal	(*a*) Normal, (*b*) underdeveloped	(*a*) Absent, (*b*) mental retardation	Chromatin $+_{ve}$, some cells 2 chromatin bodies	47	XXX
Triplo-X mosaic[16]							Chromatin $+_{ve}$, some cells 2 chromatin bodies	47/45	XXX/XO mosaic
Testicular feminization[17]	Female—sterile	Testis—undescended	Mainly male	Female	Breasts present. Pubic and axillary hair scanty. Primary amenorrhoea		Chromatin $-_{ve}$	46	XY
True hermaphrodite[18,19]	Mixed—(*a*) predominantly male, (*b*) predominantly female	(*a*) Ovary + ovotestis, (*b*) testis + ovotestis, (*c*) testis + ovary, (*d*) ? bilateral ovotestis*	Variable, both male and female differentiation. Testicular tissue ——> male differentiation on that side	Ambiguous from imperfect masculinization	Mixed male and female		Majority chromatin $+_{ve}$	46 in 5 cases who are chromatin $+_{ve}$	XX
Hermaphrodite mosaic[20]								46/45	XY/XO mosaic
Chromosomal attenuation[15]	Female	Rudimentary streaks, one of which consisted of ovarian tissue plus a few primitive follicles	Female—underdeveloped	Female—underdeveloped	Sexual infantilism	Low normal intelligence	7% cells chromatin $+_{ve}$, the chromatin body being smaller than normal	46	Xx, reduction in length of 1 X chromosome
Female pseudohermaphrodite, the adreno-genital syndrome	Female—(*a*) pseudohermaphrodite (congenital), (*b*) with virilization (adult)	Ovary	Female—(*a*) normal, (*b*) underdeveloped, (*c*) with or without prostate	Imperfect masculinization	Female with evidence of virilism	—	Chromatin $+_{ve}$	46	XX

*Recently, in our laboratory, a true hermaphrodite was shown to have bilateral ovotestes, both gonads being in the scrotum.

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the nuclear membrane, and is then usually planoconvex in shape. If it is present in a high percentage of cells, the individual is classed as chromatin positive (as in normal females). A low percentage of sex chromatin is classed as chromatin negative (as in normal males). It was then postulated that the sex chromatin results from the presence of the XX chromosomes in female nuclei.

When the test is applied to individuals with abnormal sex development it is found that:

1. In the majority of patients with Turner's syndrome who present as phenotypic females the cells are chromatin negative.
2. In Klinefelter's syndrome, although the individual is a phenotypic male, the cells are chromatin positive.
3. Patients with the testicular feminization syndrome who are phenotypic females are chromatin negative.
4. The majority of true hermaphrodites, who are phenotypically mixed male and female, are chromatin positive.

Since abnormal karyotypes had been shown to be associated with intersexes in Drosophila, it was suggested that these could occur similarly in man, and it became imperative to investigate his chromosomal constitution.

Technique of Investigation

Various tissues, e.g. bone marrow, skin, or leucocytes are cultured *in vitro* for short periods. Colchicine is introduced into the culture. This inhibits spindle formation and the cells are therefore arrested in the metaphase stage of mitosis. The addition of hypotonic sodium citrate produces swelling and divergence of the chromatids at their centromeres. The cells are then stained, e.g. with feulgen, and squash preparations are made.

Chromosomal counts are performed, and the karyotype is determined by matching the chromosomes in order of size, position of the centromere (metacentric, submetacentric or acrocentric), and length of the chromatid arms. They are accordingly arranged in pairs in 7 groups from No. 1 to No. 22 (Denver system). The X chromosome is identified as a medium-sized metacentric chromosome and the Y as a small acrocentric chromosome. In the female the sex chromosomes are XX, and in the male XY.

Correlation of Some Abnormal Sex-chromosome Karyotypes with Clinical Syndromes

Table I is a summary of some of the recent investigations of patients in whom one or other component of sex is conflicting.

The female pseudohermaphrodite of the adreno-genital syndrome has been included for clinical completeness, although no abnormal karyotype has been found. This syndrome may occur in:

(*a*) patients with adrenal cortical hyperplasia and dysfunction,

(*b*) patients with tumours of the adrenal cortex, or

(*c*) the offspring of mothers who have been receiving synthetic progesterones over a prolonged period during the first three months of pregnancy.

It is essential that this syndrome be recognized early and treated effectively.

Theory of Mechanism of Formation of the Abnormal Karyotype

A. Normal disjunction in meiosis — (Fig. 1). During parental gametogenesis the diploid number (2n) is reduced

Fig 1

to the haploid number (n) in the germ cells. The separation of like chromosomes at anaphase is referred to as disjunction. Normal ova therefore all contain one X chromosome and normal sperms either X or Y. Fertilization results in a zygote which may be either XX (a normal female) or XY (a normal male).

B. Non-disjunction of the sex chromosomes. This implies that during the first (Fig. 2) or second divisions of meiosis both sex chromosomes migrate to the same pole. This will lead to ova with XX or O chromosomes. The resultant zygote may then be one of the following combinations:

Fig 2

(*a*) XXX—triplo-X ('super-female'), (*b*) XXY—Klinefelter's syndrome, (*c*) XO—Turner's syndrome, or (*d*) YO—non-viable. Similarly, non-disjunction may take place in either meiotic division in the sperm, producing abnormal sperm karyotypes.

C. Non-disjunction in the zygote—(Fig. 3). Abnormal karyotypes may presumably also result from this phenomenon in mitosis in the zygote. If this occurs in a normal zygote, a mosaic karyotype of the order XXX/XO, i.e. triplo-X/Turner's syndrome may be produced; if in an

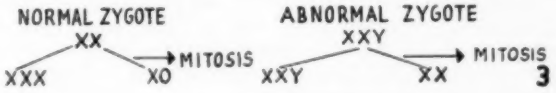

Fig 3

abnormal zygote, a mosaic XXY/XX, i.e. Klinefelter's syndrome mosaic may result.

Comparison with Lower Animals

As stated previously, abnormal karyotypes and their associated anomalies had been noted originally in Drosophila. In Table II, which compares the two species, it becomes apparent that the manifestations of the abnormal karyotypes differ, and therefore the localization of the sex genes on the chromosomes are not identical.

TABLE II. COMPARISON OF ABNORMAL KARYOTYPES IN DROSOPHILA AND MAN

Abnormal karyotype	*Drosophila*	*Homo sapiens*
XXY	Fertile, apparently normal female	Sterile male
XO	Sterile, apparently normal male	Sterile female
XXX	Super-female with accentuated secondary sex characters	(*a*) Sterile female, (*b*) fertile female, (*c*) mental defects
YO	Non-viable	Non-viable

*Recently, in our laboratory, a true hermaphrodite was shown to have bilateral ovotestes, both gonads being in the scrotum.

Recently, studies in the mouse have shown the occurrence of the XO karyotype and also the presence of masculinizing genes on the Y chromosome.

DISCUSSION

Investigation of the chromosomal constitution in man has shown a correlation between the karyotype and the nuclear chromatin pattern. The presence of the sex chromatin body in the interphase nucleus indicates XX chromosomes. Double sex chromatin bodies suggest at least XXX chromosomes, e.g. triplo-X, 'super-female'. The explanation of the formation of the sex chromatin is unknown. A theory has been proposed that the X chromosomes display differential behaviour and that it is the heteropyknotic X chromosome that accounts for the sex chromatin. Nuclear sex determination is an important laboratory procedure in patients who present with clinical syndromes involving sexual development. In chromatin-negative individuals with Turner's syndrome it can be deduced that the karyotype is XO and in chromatin-positive patients with Klinefelter's syndrome the karyotype is XXY.

Abnormal sex differentiation may occur despite apparently normal sex determination. In the testicular feminization syndrome the karyotype is male, the gonads are testes, but the phenotype is female. In this condition the testes may be physiologically abnormal. Similarly, in the syndrome of pure gonadal dysgenesis, the karyotype is XY, but the gonad remains in an undifferentiated state and the phenotype is female. Perhaps in both these syndromes the individual target organs do not respond to their respective inductors. Conversely, in 5 true hermaphrodites the karyotype was found to be XX, in spite of which testicular differentiation occurred with accompanying male differentiation on the corresponding side.* The possibility of translocation of a portion of the Y chromosome bearing masculinizing genes must be considered here.

Abnormal sex determination is usually, but not inevitably, accompanied by abnormal sex differentiation, e.g. Turner's syndrome and Klinefelter's syndrome. However, there is a recorded case in which the karyotype is XO with completely normal sexual development.[21]

The rôle of the sex chromosomes is therefore incompletely understood. Certain deductions may, however, be drawn:

1. In man the Y chromosome carries masculinizing genes (cf. Drosophila in which the Y appears to be inert). Its presence is linked with the development of the testis.

2. Testicular development may occur in the absence of the Y chromosome.

3. The Y chromosome does not ensure the development of a testis.

4. XX chromosomes are not essential for normal female sex development.

5. The sex chromosomes are not solely responsible for sexual development in a given direction, and there are probably masculinizing and feminizing genes on the autosomes. Coordination of both may be required for normal sex development.

6. In the presence of vestigial gonads female development invariably occurs.

* In a recent report[22] a boy of 8 who is a phenotypic male was shown to have an XO chromosomal constitution and a rudimentary testis. This provides further evidence of testicular differentiation in the absence of the Y chromosome.

SUMMARY

1. Sexual development depends on the process of sex determination and sex differentiation.

2. The sex of the individual should be assessed at different levels.

3. Nuclear sex determination is an important laboratory investigation in patients with abnormal sex development. The mode of the formation of the sex chromatin is unknown.

4. Abnormal sex-chromosome karyotypes may be associated with certain clinical syndromes.

5. Abnormal karyotypes are at present explained on non-disjunction of chromosomes at cell division.

6. The rôle of the sex chromosomes in sex development is incompletely understood, but the Y chromosome appears to be more important than was previously suspected. The possibility of autosomal sex determination in conjunction with the sex chromosomes must be postulated.

7. Abnormal sexual development can occur at any stage of differentiation. An abnormal sex-chromosome karyotype is only one of the known factors which is associated with, and may account for, sexual anomalies.

8. Several factors in sexual differentiation remain to be evaluated. These include the response of target organs to inductors, the possibility of abnormal physiologic function of the gonad, and the possible influence of the germ cells on sex-organ differentiation and behaviour.

REFERENCES

1. (*a*) Tjio, J. H. and Levan, A. (1956): Hereditas (Lund.), **42**, 1.
(*b*) Ford, C. E. and Hamerton, J. L. (1956): Nature (Lond.), **178**, 1020.
2. Jost, A. (1953): Recent Progr. Hormone Res., **8**, 379.
3. Barr, M. L. and Bertram, E. G. (1949): Nature (Lond.), **163**, 676.
4. Jacobs, P. A. and Strong, J. A. (1959): *Ibid.*, **183**, 302.
5. Harnden, D. G. (1960): Brit. J. Exp. Path., **41**, 31.
6. Muldal, S. and Ockey, C. H. (1960): Lancet, **2**, 492.
7. Ford, C. E., Polani, P. E., Briggs, J. H. and Bishop, P. M. F. (1959): Nature (Lond.), **183**, 1030.
8. Crooke, A. C. and Hayward, M. D. (1960): Lancet, **1**, 1198.
9. Ferguson-Smith, M. A., Johnston, A. W. and Handmaker, S. D. (1960): *Ibid.*, **2**, 184.
10. Ford, C. E., Jones, K. W., Polani, P. E., de Almeida, J. C. and Briggs, J. H. (1959): *Ibid.*, **1**, 711.
11. Fraccaro, M., Kaijser, K. and Lindsten, J. (1959): *Ibid.*, **1**, 886.
12. Harnden, D. G. and Stewart, J. S. S. (1959): Brit. Med. J., **2**, 1285.
13. Ford, C. E. (1960): Amer. J. Hum. Genet., **12**, 104.
14. Jacobs, P. A., Baikie, A. G., Court Brown, W. M., MacGregor, T. N., MacLean, N. and Harnden, D. G. (1959): Lancet, **2**, 423.
15. Fraser, J. H., Campbell, J., MacGillivray, R. C., Boyd, E. and Lennox, B. (1960): *Ibid.*, **2**, 626.
16. Jacobs, P. A., Harnden, D. G., Court Brown, W. M., Goldstein, J., Close, H. G., MacGregor, T. N., MacLean, N. and Strong, J. A. (1960): *Ibid.*, **1**, 1213.
17. Jacobs, P. A., Baikie, A. G., Court Brown, W. M., Forrest, H., Roy, J. R., Stewart, J. S. S. and Lennox, B. (1959): *Ibid.*, **2**, 591.
18. Harnden, D. G. and Armstrong, C. N. (1959): Brit. Med. J., **2**, 1287.
19. Ferguson-Smith, M. A., Johnston, A. W. and Weinberg, A. N. (1960): Lancet, **2**, 126.
20. Hirschhorn, K., Decker, W. H. and Cooper, H. L. (1960): *Ibid.*, **2**, 319.
21. Bahner, F., Schwartz, G., Harnden, D. G., Jacobs, P. A., Hienz, H. A. and Walter, K. (1960): *Ibid.*, **2**, 100.
22. Bloise, W., de Assis, L. M., Bottura, C. and Ferrari, I. (1960): *Ibid.*, **2**, 1059.

South African Medical Journal : Suid-Afrikaanse Tydskrif vir Geneeskunde

EDITORIAL : VAN DIE REDAKSIE

MALNUTRITION IN INFANTS AND YOUNG CHILDREN

One of the most alarming problems in the practice of medicine is the fact that there are such a great number of treatable and preventable conditions which remain outside the scope of effective therapy because of the unsatisfactory social and economic background of these problems.

One striking example of a condition of this nature is the problem of malnutrition in children. As is evident from reports which have been published in the medical press, it becomes increasingly clear that there are many thousands of infants and young children in South Africa today who are doomed to defective mental and physical development purely because they are receiving insufficient nourishing food.

Many children die from frank nutritional disorders such as marasmus, kwashiorkor, or rickets, and many from gastro-enteritis and pneumonia, which should not be fatal (particularly in the 1 - 4 years age group), but which are the cause of thousands of non-European deaths in childhood below the age of 5 years. It is a point of great significance that, while the infantile mortality rate among non-European infants is 4 - 6 times the rate among European infants, the death rate in the age group 1 - 4 among the Coloured population is 15 times, and among the Bantu, 30 times as great as the European death rate at this age. A large proportion of these deaths is caused by the conditions mentioned earlier. The basic reason for this distressing fact lies in lack of essential foodstuffs, particularly protein, in the daily diet of these children.

Recently, Robertson and her colleagues[1] pointed out, from the available figures, what an enormous loss of infant life there is in this country from gastro-enteritis, and they produced evidence to show that a very large proportion of this is caused by malnutrition.

Another revealing article was published by Moodie.[2] She studied the background of 150 hospitalized children with severe kwashiorkor, using the patients' siblings as controls. Her studies indicate that this disease tends to occur among child populations that show widespread evidence of growth retardation.

She also pointed out that one of the features that differentiates kwashiorkor, as it occurs in Cape Town, from the syndrome as it occurs in most communities in Africa, seems to be the earlier introduction of artificial feeding with its hazards of infection and overdilution. This is the case with both Cape Coloured and African mothers who, in this respect, resemble the Indian and Negro mothers in Trinidad.

Other pertinent findings noted by Moodie are dietary deficiency associated with poverty, ignorance, prevalence of infection, and a general state of poor housing and social disorganization among unskilled labourers in an area of rapid industrial development.

In yet another recent article, which was published in this *Journal*,[3] Snyman and Murray describe their findings in a study of 964 non-European children who were seen in the outpatient and casualty departments of the Karl Bremer Hospital, Bellville, Cape Town, during May 1960.

It was found that at least 28·3% of these infants received either no milk or less than 1 pint per day. Between the ages of 1 and 2 years, 66% received either no milk or less than 1 pint per day. Of those children between the ages of 6 months and 12 years, only 10·9% were of 'textbook' average normal weight or heavier ; 48% were down to 24·9% below this average normal weight ; and 41·1% were 25% below or worse. An analysis of the diagnoses confirmed the extremely poor state of nutrition of these patients. Kwashiorkor or nutritional marasmus was present in 8·4%, and 48·4% had some condition usually associated with malnutrition or undernutrition, e.g. rickets, tuberculosis, gastro-enteritis, or bronchopneumonia.

These findings assume even more shocking proportions if we view them against the background of the work done by Scrimshaw and Béhar,[4] who have recently pointed out that for every case of kwashiorkor in a population group, there are at least a hundred cases of underlying protein malnutrition — sometimes referred to as pre-kwashiorkor. These cases can best be recognized by the fact that their weight is below that expected for their age.

A great deal can be done to prevent this saddening and deplorable state of affairs. A step in the right direction would be to make available inexpensive milk and other sources of protein for pre-school children. But here, as elsewhere, we cannot disregard the factors of education and guidance. Moodie,[2] to whose work we have already referred, draws attention to a disturbing aspect of this problem, i.e. that so large a part of the Coloured population does not attend clinics. There is an understandable tendency to consider that the mothers are too idle to do so, but this makes no allowance for such factors as the distance from clinics, the time required to be absent from the home and other children, the failure to understand why a 'well' child should go to a clinic, the inability to pay for milk, the fear of reproof if money is not forthcoming, and the apathy engendered by prolonged poor nutrition, fatigue, slum living, ignorance, and limited intellectual ability. All these factors need to be studied thoroughly.

The whole problem of malnutrition in infants and young children will have to be approached with an open mind and with reference to all its medical and social implications. This approach, if successfully carried out, will diminish the necessity for expensive new hospitals, since many of the sick and undernourished children should

never have been allowed to deteriorate to a condition requiring prolonged hospitalization. The health structure of a community is as satisfactory or unsatisfactory as the foundations on which it rests. The care of the health of the children of all sections of the community is a social and moral responsibility which we dare not evade. This problem can only be solved on the basis of purposeful and imaginative planning.

1. Robertson, I., Hansen, J. D. L. and Moodie, A. (1960): S. Afr. Med. J., **34**, 338.
2. Moodie, A. (1961): J. Pediat., **58**, 392.
3. Snyman, J. D. and Murray, A. B. (1961): S. Afr. Med. J., **35**, 595.
4. Scrimshaw, N. S. and Béhar, M. (1959): Fed. Proc., **18**, 82.

NARKOTISEUR EN / OF GENEESHEER

Gedurende die afgelope vyf-en-twintig jaar het anestesiologie as 'n spesialiteitsfeer van krag tot krag ontwikkel sodat dit vandag 'n eerbare plek vul in die bolwerk van die mediese praktyk. Die eer vir hierdie uittog uit 'n voormalige woestyn is hoofsaaklik te danke aan 'n handjievol alombekende vakmanne in die Britse en Amerikaanse narkosepraktyk. Hulle konserwatiewe en weldeurdagte leiding behels nog steeds die kern van ontwikkeling en bied 'n gesonde lees waarop die toekoms van hierdie jong spesialiteit geskoei kan word. Belangrik is dit om daarop te let dat hierdie baanbrekers in die eerste plek geskoolde klinici in die praktyk van die interne medisyne is.

Dit is tot 'n mate te betreur dat tegniese aspekte voortdurend benadruk word wanneer die ontwikkelingsgang van die narkosepraktyk in oënskou geneem word. Tegniese vaardigheid is geensins die enigste kenmerk van 'n goeie narkotiseur nie, net so min as wat bloot operatiewe vaardigheid alleen 'n uitmuntende chirurg kenmerk. Hunter[1] het reeds tien jaar gelede voorspel dat die tyd sal aanbreek wanneer die status van die narkotiseur beoordeel sal word op grond van die kwaliteit van sy kliniese insig in teenstelling met bloot tegniese vaardigheid.

Nog meer betreurenswaardig is dit dat so min, indien enige, pasiënte die narkotiseur as 'n integrale deel van die sukses van hulle chirurgiese ondervinding beskou. Die pasiënt se verhouding tot sy narkotiseur is in die reël die mees onpersoonlike aspek van sy verblyf in die hospitaal.[2] Heel uitsonderlik gebeur dit dat die ideale vereiste van ons primêre doelwit, naamlik 'n kerngesonde geneesheer-pasiënt verwantskap, vervul word.

Om hierdie ideale verwantskap aan te knoop sal in die toekoms 'n opdraende taak wees, want tot ons leed word die omvang van narkosepraktyk op die huidige tydstip nog grootliks net met die operasiekamer vereenselwig. Die vrugbare kliniese veld van die pre- en postoperatiewe periode lê braak en word al te dikwels stiefmoederlik benut. Vry algemeen nog vind die eerste kennismaking tussen pasiënt en narkotiseur in die operasiekamer plaas en eindig wanneer die tegniese fase van die chirurgiese ingreep voltooi is; moontlik nog voor die pasiënt volle bewussyn herwin het.

Die huidige ontwikkelingsrigting met betrekking tot vereistes vir spesialisasie toon 'n neiging om steeds meer en meer die kwalifikasie van ondervinding in die algemene praktyk op die agtergrond te skuif. Die gevaar bestaan dus dat 'n geslag narkotiseurs ontluik wat geen, of uiters geringe, kontak het met die probleme van mediese praktyk in sy breedste vorm, en wat volgens die huidige aard van hulle spesialiteitspraktyk weinig geleentheid het vir die ideale persoonlike kontak met hulle pasiënte; of die vrugbare geleenthede wat wel bestaan nie genoegsaam benut nie.

Die wind van verandering waai deesdae om elke hoek, en as die narkotiseur nie bloot as vaardige farmaseutiese tegnikus oor die bewustelose indiwidu wil uitwarrel nie, behoort hy opnuut die geleentheid te soek om terug te keer tot 'n verhouding met sy pasiënte wat in die eerste plek dié van geneesheer in die breedste sin van die woord is. Die benamings goeie geneesheer en vaardige spesialiteit is nie noodwendig sinoniem nie. In hierdie opsig rus daar 'n swaar verantwoordelikheid op die skouers van diegene wat gemoeid is met die onderrig van aspirant narkotiseurs om te verhoed dat die pasiënt as mens met alledaagse drange, gevoelens, en bekommernisse nie in die doolhof van tegniek en statistiese gegewens verlore raak nie.

1. Hunter, A. R. (1952): Anesthesiology, **13**, 108.
2. Karp, M. (1957): Curr. Res. Anesth., **36**, 36.

DIVERTICULAR DISEASE OF THE COLON

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PART III

THE RADIOLOGICAL DIAGNOSIS OF DIVERTICULITIS

As was expected, the literature on the radiological diagnosis of diverticulitis is replete with confusing and contradictory statements. Demonstration of diverticula of the colon is reasonably simple either on barium-enema or barium-meal examination, but contradictory statements are rife when it comes to the criteria for diagnosing the presence of complicating inflammation. This is one of the main reasons why estimates of the incidence of this complication are so divergent.

Some of the pioneer work on the radiology of this condition was done by Spriggs and Marxer[40-42] from 1925 to 1929, and the stages in the development of the condition as postulated by them are still widely accepted: (1) The prediverticular stage, (2) established diverticulosis, and (3) diverticulitis.

The prediverticular stage is said to be recognized by 'fine spastic notches either grouped on the summits of the haustral bulges, or replacing these bulges'. Fig. 7 is a diagrammatic representation of this stage after Spriggs and Marxer, and the transverse colon in Fig. 8 could be considered to show the features of 'prediverticulosis'. The notches are claimed to be

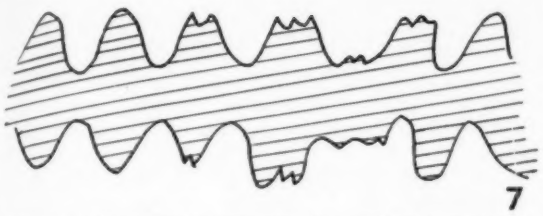


Fig. 7. Diagrammatic representation of 'prediverticulosis' (after Spriggs and Marxer).

incipient hernial pouches of colonic mucosa through the muscularis, and it is alleged that follow-up of these cases will

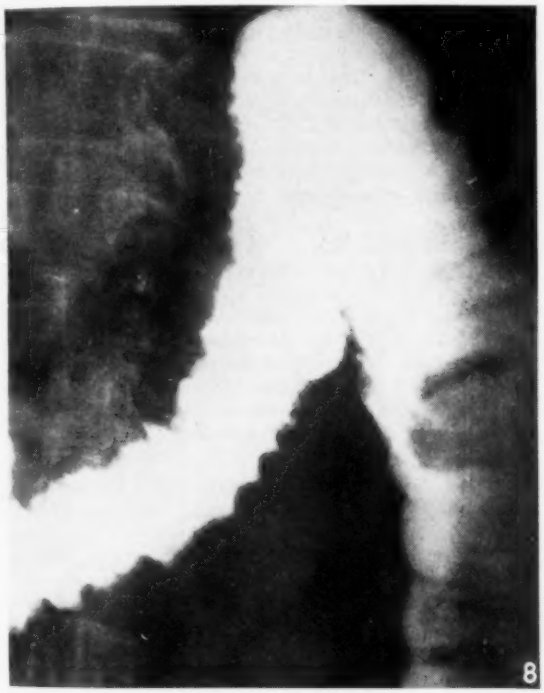


Fig. 8. Rippling of the transverse colon. Prediverticulosis?

reveal the development of true diverticula *at these sites*. This concept has been supported by Lockhart-Mummery and Hodgson,[43] and more recently Henderson,[17] writing from St. Mark's Hospital, has also accepted and even elaborated it.

It is significant that Spriggs and Marxer, and also Henderson, considered the aetiology of diverticulosis to be inflammation, in that antecedent inflammatory episodes cause the weak areas in the bowel wall. This view commands little if any support today. Todd,[3] also writing from St. Mark's, in 1955, ascribed the rippling to spasm of the muscularis mucosae from the irritation of inflammation complicating established diverticulosis. He also described a smooth waviness of the mucosa from mucosal oedema. Evidence that Todd's view may be nearer the truth is found in the observation that this appearance is most common in the sigmoid area and that it is frequently found in association with established diverticula situated more proximally.

It is well known that diverticulosis almost invariably starts in the sigmoid area. It is in fact the sole area affected in 47% of cases.[19] To call this appearance in the sigmoid area 'prediverticulosis', while the proximal colon, which is almost invariably involved at a later stage chronologically, is the seat of established diverticulosis, does not fit in with the facts observed. The sigmoid is also almost invariably the site of complicating diverticulitis, so that it may be reasonable to ascribe these radiological appearances to inflammation. It will be shown later that the acceptance of this explanation may be too facile, since these features may also merely be the manifestation of the normal irritability of the colon during barium-enema examination. The concept of Spriggs and Marxer of a *radiological* prediverticular stage does therefore not appear to be a correct one.

With regard to *diverticulitis*, the 'saw-tooth' deformity, as depicted diagrammatically in Fig. 9, is widely held to be the

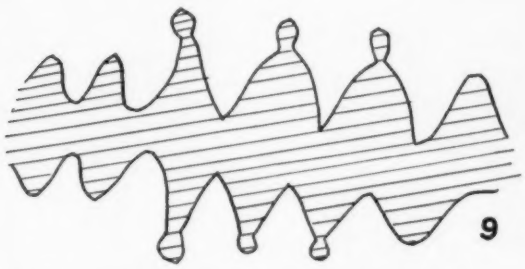
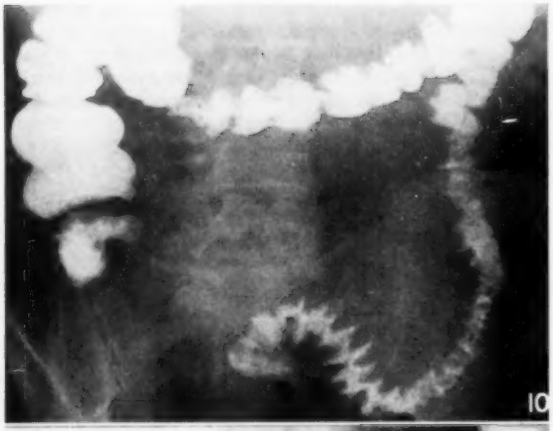


Fig. 9. Diagrammatic representation of 'saw-toothing'.

most important radiological feature. Fig. 10 is a good example and occurred in a woman with unequivocal diverticulitis. There is a difference of opinion concerning the cause of this appearance. The usual view[3] is that it is caused by spasm of the circular muscle of the bowel associated with oedema of the haustral folds. Goulard and Hampton,[44] however, felt that it is caused by a fibrotic shortening of the bowel and thickening of the folds. The deformity, they say, is permanent and indicative of previous inflammation. As evidence of this statement they found that 10 of their patients had no change in the deformity when observed at intervals of 3 months - 3 years. Furthermore, of 45 patients with a demonstrable saw-tooth deformity, only 20 had clinical acute diverticulitis, and conversely, 15 patients (43%) with clinical acute diverticulitis had no saw-tooth deformity. In the present series there is evidence that both views may be correct.

Fig. 11 is another film of the patient depicted in Fig. 10, and was taken at the same examination. The saw-tooth appearance has clearly been transient and cannot result from fibrotic deformity. Figs. 12, 13, and 14 are films of another patient taken at monthly intervals after the establishment of a transverse colostomy for a perforated diverticulum. At the time the X-rays were taken there was no residual evidence, locally or generally, of acute inflammation, so that it appears to be more reasonable to ascribe the radiological features in this case to a fixed deformity than to spasm.

With regard to spasm, a major difficulty is the differentiation between pathological spasm from inflammatory irritability, and the transitory irregular contractions



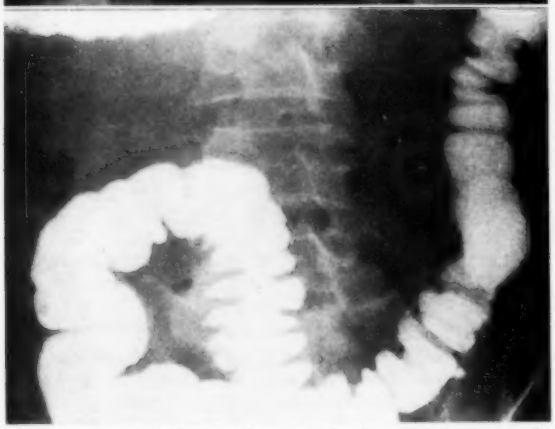


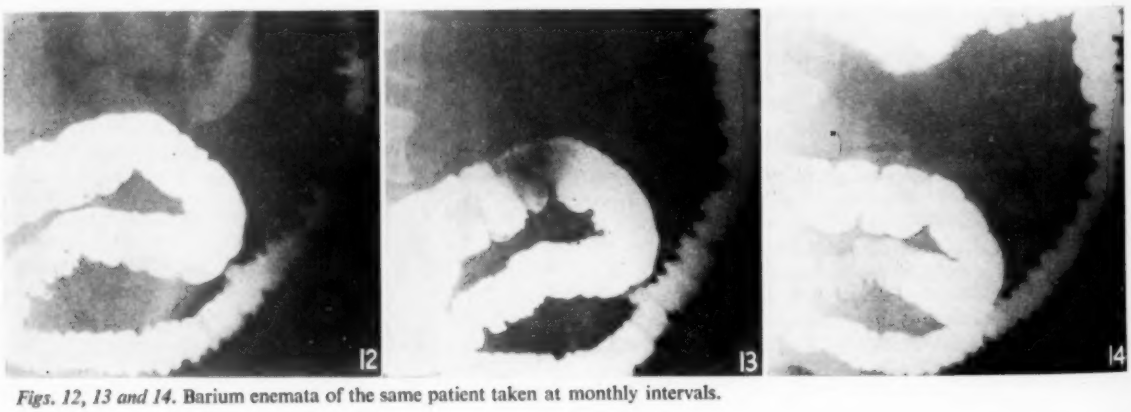
Fig. 10. 'Saw-toothing' of the sigmoid colon. This patient had unequivocal clinical diverticulitis.

Fig. 11. Barium enema of the same patient as in Fig. 10 taken at same examination. The saw-toothing has disappeared.

of the colon so frequently seen during filling. Goulard and Hampton[44] have defined spasm, as opposed to this normal irritability, as a state of contraction persisting throughout any one examination, and that it is best assessed with the remainder of the colon filled and the patient lying prone. It is significant, however, that of 45 of their patients with demonstrable 'pathological' spasm, only 20 had clinical acute diverticulitis, and conversely, 15 patients with clinical acute diverticulitis, often with demonstrable abscesses, did not manifest any spasm at all. The experience of this series provides many examples to support them. For instance, Figs. 10 and 11 demonstrate spasm ('saw-toothing') which was not persistent during the examination, and yet the patient had undoubted diverticulitis (it must be pointed out, however, that the barium-enema examination was not performed during the course of the acute illness). Conversely, Fig. 15 shows the filling of a pericolic abscess and yet there is no spasm at all in the adjacent colon, and were it not for the filling of the cavity, the radiological diagnosis would certainly have been diverticulosis only. Figs. 16 and 17 were obtained during a single examination of a patient in whom there was no clinical suspicion of past or present diverticulitis. (It was a routine barium enema in a case of haemorrhoids.) Fig. 16 may be held to show the features of prediverticulosis, diverticulosis and diverticulitis, but Fig. 17, which is another film during the same examination, and the clinical picture, are evidence that the appearances are merely caused by 'normal irritability'. The correlation between spasm and the clinical picture is thus very poor.

It is possible that this irritability, while not necessarily present in or caused by inflammation, and which is more commonly seen in the sigmoid area than elsewhere in the large bowel, may be a manifestation of some disorder of the motor activity of the colon, and that it may be important in the pathogenesis of diverticulum formation. Further research is being done on this aspect. Superficially this view seems to be a return to the prediverticulosis concept of Spriggs and Marxer, but the fundamental difference should be quite apparent.

Attention should also be paid to the observation of Wolf, Khilnani and Marshak[45] that an appearance similar to 'saw-toothing', namely, marked haustral asymmetry, is in fact an established part of established *diverticulosis* (Fig. 18). As a diverticulum extends laterally, there is frequently sacculation of an involved haustrum. The damaged muscular fibres bunch, 'as when an elastic band is cut or weakened in one place' producing thick interhaustral plicae. The important feature is the unusual distensibility of the bowel.



Figs. 12, 13 and 14. Barium enemata of the same patient taken at monthly intervals.

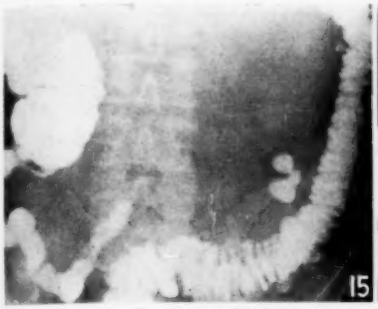


Fig. 15. Peri-colic abscess filled. No spasm in adjacent colon.

Fig. 16. Rippling of 'prediverticulosis'? Saw-toothing of diverticulitis?

Fig. 17. Same patient and same examination as depicted in Fig. 16. The spasm producing the rippling has been transient. Clinically there was no past or present evidence of diverticulitis.

Fig. 18. Marked asymmetry and sacculation of haustra. Established diverticulosis.

A fact not widely appreciated is that the barium-enema examination may be quite normal in acute diverticulitis, since the diverticulum may fail to fill owing to obstruction of its neck. Six of Goulard and Hampton's[44] cases showed no demonstrable diverticula at all. Four of these had demonstrable abscesses, and in one case a barium enema immediately before operation was perfectly normal.

The presence of fixed anatomical changes, owing to the presence of the intramural but extramucosal inflammatory mass, provides reliable radiological confirmation of the diagnosis of diverticulitis. There may be a well-defined filling defect (Fig. 5 and 6), a poorly demarcated area of induration (Fig. 19), or an abscess cavity (Figs. 15 and 20), but, clearly, the absence of these features by no means excludes the diagnosis.

We may then conclude that:

1. The barium enema may be quite normal in diverticulitis.

2. The barium enema may show only the classical features of diverticulosis in a proved case of diverticulitis.

3. The interpretation of spasm is difficult and requires further research. Diverticulitis may occur in the absence of any radiological evidence of spasm, and *vice versa*.

4. Fixed anatomical defects provide reliable radiological confirmation of the diagnosis of diverticulitis, but their absence does not exclude the diagnosis.

5. The clinical aspect of the diagnosis is the most reliable in the present state of our knowledge.

There is a paucity of radiological observations made during the acute phase of the disease. The hazards of this procedure have probably been overstated,[44] and pursuance of this line of investigation may fill a gap in our knowledge.

MANAGEMENT

The immediate management of the complications of diverticulitis has been discussed under the relevant sections. It remains only to discuss the rôle of elective surgery in this condition.

The orthodox view[6] has been that the only surgery of diverticulitis is the surgery of its complications, and such surgery as was necessary was usually staged. In recent years 2 trends are discernible. Firstly, there has been an increasing tendency to employ single-stage procedures,[13,24,25,29] even in the presence of severe complications. Secondly, many authorities have recommended earlier surgery in an attempt to forestall complications.[24,29]

A word of warning should be sounded with regard to this more aggressive attitude, and a plea is made for judgement on the merits of each case individually. This view obtains support from Friesen and Schmidt[35] who state that no condition in general surgery requires more seasoned judgement if prolonged morbidity and even mortality are to be kept at a minimum.

It is apparent from our series that 2 very serious complications, namely, massive haemorrhage and rupture of a diverticulum, tend to occur quite unheralded. In fact, they are

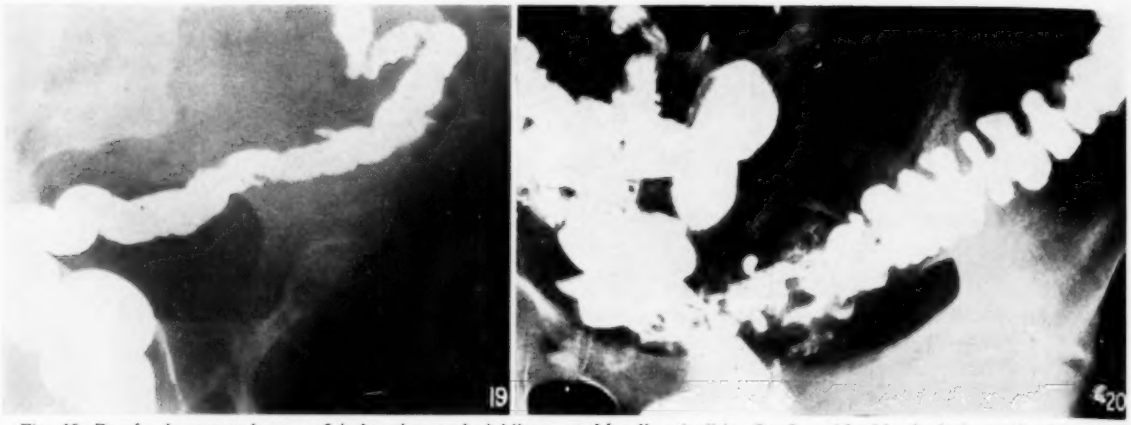


Fig. 19. Poorly demarcated area of induration and rigidity caused by diverticulitis. Confirmed by histological examination after resection.

Fig. 20. Barium filling a pericolic abscess cavity.

essentially complications of diverticulosis, and a past history to suggest episodes of diverticulitis is usually not forthcoming. A programme of prophylactic surgery to forestall such complications is patently ludicrous, in view of the great incidence of diverticula, especially in the older age groups. As we have stated earlier, a very reasonable case can even be made out for not subjecting patients who have merely ruptured a diverticulum to any excisional surgery at all, and in the case of massive haemorrhage the good results from a conservative approach, combined with the difficulty of localizing the bleeding point at operation, make this conservative approach a reasonable one.

With regard to the place of surgery in patients with recurrent episodes of acute diverticulitis, a reasonable attitude is to consider resection after the second attack has settled, but a plea is likewise made to suit therapy to the needs of the individual patients. Excisional surgery for diverticulitis coli is difficult at the best of times, and obesity, to which a fair proportion of these people are prone, adds considerably to the hazards of colonic surgery. We would agree with Welch and Rodkey,[46] from the Massachusetts General Hospital, who, while acknowledging the place of 1-stage resections in this condition, emphasize the necessity for judgement and conservatism in selecting the type of surgery. They point out that the surgeon is not operating for carcinoma, that most complications after surgery are referable to the anastomotic line, and that a temporary colostomy is a small price to pay for the added safety. 'Rigid standardization should be avoided, and cognizance should be taken of a multiplicity of local and general factors'.

Lloyd-Davies[1] has also stated as recently as 1955 that there will always be a group of irresectable cases, either because of age or poor risk, or because colostomy has failed to control the local infection. He cites a case where a vesicocolic fistula appeared 7 months after the performance of a colostomy. One patient in this series had a transverse colostomy only because of cardiac contraindications to a major resection, and in a further patient who also has a colostomy, resection has been repeatedly put off for several years because she was unwilling to reduce her enormous obesity. Difficulty in excluding carcinoma (there were 4 such cases in this series), certainly warrants the more aggressive attitude.

SUMMARY

1. Ninety patients with symptomatic diverticular disease of the colon from the Johannesburg General Hospital are reviewed.

2. The anatomy and pathogenesis of diverticula are discussed. The rôle of the anatomical relations of the blood vessels to the bowel in causing potential weaknesses in the wall at the sites of their penetration is well established, but the precipitating factors determining the appearance of diverticula in the individual person are still obscure.

3. Massive haemorrhage is almost invariably a complication of diverticulosis and not diverticulitis. A conservative attitude to this complication is justified by the results obtained with it, and by the considerable difficulties facing the surgeon if he has to operate. Diverticulitis seldom bleeds massively, and the definitive treatment in these cases is determined by other considerations.

4. A spreading or generalized peritonitis may result from the rupture of a diverticulum with little or no evidence of diverticulitis, rupture of a pericolic abscess, or extension of the acute inflammatory process without evidence of free perforation or rupture of an abscess. The mortality is very high. Consideration of the fatal cases produces evidence that vigorous and adequate restoration of blood volume in these patients is the most important single factor to be considered if the mortality rate is to be decreased. Prevention of continued re-infection of the peritoneal cavity must also be achieved. There is much difference of opinion regarding the surgical measures which may best accomplish this.

5. In cases presenting an abdominal or pelvic mass, the differentiation from carcinoma is important and may be difficult. The criteria available for differential diagnosis are evaluated.

6. Fistula formation has become a rare complication of diverticulitis. The more efficient management of the acute

attack made possible by the advent of antibiotics is probably the most important reason for this.

7. It is important to appreciate that there are at least 3 possible mechanisms whereby intestinal obstruction can complicate diverticulitis. A case illustrating each is presented.

8. The radiological criteria for the differential diagnosis between diverticulosis and diverticulitis are discussed. Attention is drawn to several widespread misconceptions, and the importance of considering the clinical manifestations in conjunction with the radiological features is emphasized. The effect of an accurate differentiation on the assessment of the natural history of the condition, and therefore on a reasonable therapeutic attitude to the condition, is discussed.

9. While cognizance is taken of the possible advantages of a more aggressive attitude to this condition, a plea is made for avoiding a rigidly standardized therapeutic approach to the indications for surgery and the type of surgery employed.

I am indebted to Prof. D. J. du Plessis for invaluable help and criticism; to Dr. J. S. Enslin, Deputy Superintendent of the Johannesburg General Hospital, for permission to publish these cases; to surgeons on the staff of the Johannesburg General Hospital for access to their case records; to Dr. M. Berk of the Department of Radiology for helpful advice; and to Mr. D. Smith of the Department of Surgery for preparation of the photographs.

REFERENCES

1. Lloyd-Davies, O. V. (1953): Proc. Roy. Soc. Med., **46**, 407.
2. Welch, C. F., Allen, A. W. and Donaldson, G. A. (1953): Ann. Surg., **138**, 332.
3. Todd, I. P. (1955): Ann. Roy. Coll. Surg. Engl., **16**, 118.
4. Morton, J. J. (1946): Ann. Surg., **124**, 725.
5. Ochsner, H. C. and Bargen, J. A. (1935): Ann. Intern. Med., **9**, 282.
6. Pemberton, J. de J., Black, B. M. and Maino, C. R. (1947): Surg. Gynec. Obstet., **85**, 523.
7. Grout, J. L. A. (1949): Brit. J. Radiol., **22**, 442.
8. Smithwick, R. H. (1942): Ann. Surg., **115**, 669.
9. Bacon, H. E. and Sherman, L. F. (1950): Amer. J. Surg., **80**, 3.
10. Edwards, H. C. (1954): Ann. Roy. Coll. Surg. Engl., **14**, 371.
11. Lockhart-Mummery, J. P. (1938): Lancet, **2**, 1401.
12. Ford, H. S. (1953): Canad. Med. Assoc. J., **69**, 506.
13. Boyden, A. M. (1950): Ann. Surg., **122**, 94.
14. McGowan, F. J. and Wolf, W. I. (1952): Gastroenterology, **21**, 119.
15. Drummond, H. (1916 - 17): Brit. J. Surg., **4**, 407.
16. Fausler, W. A. (1940): Trans. Amer. Proctol. Soc., **41**, 231.
17. Henderson, H. P. (1944): Brit. J. Radiol., **17**, 197.
18. Rankin, F. W. and Graham, A. S. (1939): *Carcinoma of the Colon and Rectum*, p. 117. London: Baillière, Tindall and Cox.
19. Horner, J. L. (1958): Amer. J. Dig. Dis., **3**, 343.
20. Fraenkel, G. J. (1954): Brit. J. Surg., **41**, 643.
21. Hoar, C. S. and Bernhard, W. F. (1954): Surg. Gynec. Obstet., **99**, 101.
22. MacLaren, I. F. (1957): J. Roy. Coll. Surg. Edin., **3**, 129.
23. Maingot, R. (1955): *Abdominal Operations*, 3rd ed. London: Lewis.
24. Bacon, H. E. and Valiente, M. A. (1956): Amer. J. Surg., **91**, 178.
25. Ryan, P. (1958): Brit. J. Surg., **45**, 611.
26. Guy, C. C. and Werelius, C. Y. (1952): Surg. Clin. N. Amer., **32**, 91.
27. Rankin, F. W. and Brown, P. W. (1930): Surg. Gynec. Obstet., **50**, 836.
28. Colcock, B. P. and Sass, R. E. (1954): *Ibid.*, **99**, 627.
29. Waugh, L. J. M. and Walt, A. J. (1957): *Ibid.*, **104**, 690.
30. Laufman, H. (1941): Int. Abstr. Surg., **73**, 222.
31. Arnheim, E. E. (1940): Ann. Surg., **112**, 352.
32. Buie, L. A. and Jackman, R. J. (1943): J. Amer. Med. Assoc., **121**, 1144.
33. Smith, N. D. (1951): Amer. J. Surg., **82**, 583.
34. Schatzki, R. (1940): Radiology, **34**, 651.
35. Friesen, L. and Schmidt, E. R. (1955): Surg. Clin. N. Amer., **35**, 1385.
36. Telling, W. H. M. and Gruner, O. C. (1917): Brit. J. Surg., **4**, 468.
37. Mayo, C. W. and Blunt, C. B. (1950): Surg. Gynec. Obstet., **91**, 612.
38. Reid, S. E. and Workman, C. M. (1955): Quart. Bull. Northw. Univ. Med. Sch., **29**, 190.
39. Ormond, J. K. (1949): J. Urol. (Baltimore), **61**, 554.
40. Spriggs, E. L. and Marxer, O. A. (1927): Lancet, **1**, 1067.
41. *Idem* (1925): Quart. J. Med., **19**, 1.
42. Spriggs, E. L. (1929): Brit. Med. J., **2**, 566.
43. Lockhart-Mummery, J. P. and Hodgson, H. G. (1931): *Ibid.*, **1**, 525.
44. Goulard, A. and Hampton, A. O. (1954): Amer. J. Roentgenol., **72**, 213.
45. Wolf, B. S., Khilnani, M. and Marshak, R. H. (1958): *Ibid.*, **77**, 726.
46. Welch, C. F. and Rodkey, G. V. (1956): Surgery, **39**, 712.
47. Stone, H. B. (1946): Ann. Surg., **124**, 743.
48. *Idem* (1944): *Ibid.*, **120**, 582.
49. Jensen, G. L. (1958): Amer. J. Surg., **95**, 813.
50. Foster, R. L. and Fisher, R. F. (1954): Amer. Surg., **20**, 734.
51. Kunath, G. A. (1956): Amer. J. Surg., **91**, 911.
52. du Plessis, D. J. (1960): Personal communication.
53. Gouverneur, R., Soupault, R. and Latifi, M. (1938): J. Chir. (Paris), **51**, 215.
54. Lynn, T. E., Farrell, J. I. and Grier, J. P. (1958): Surgery, **76**, 956.
55. Ewell, G. H. (1951): Amer. J. Surg., **82**, 597.
56. Edwards, H. C. (1954): *Recent Advances in Surgery*, 4th ed., p. 213. London: J. and A. Churchill.

GEOGRAPHICAL AND RACIAL DISTRIBUTION OF DIPHTHERIA IN SOUTH AFRICA

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Until World War II South Africa enjoyed a low diphtheria rate compared with other countries.[1-3] Since then, the disease has been suppressed or even eliminated in many countries, whereas the last survey in South Africa (1952)[3] showed that the annual incidence here had remained unchanged.

The purpose of this paper is to bring up to date the national picture of diphtheria and to examine the geographical and racial distribution of the disease.

MATERIAL AND METHODS

Demographic figures for the period before 1958 were extracted from the annual reports of the Department of Health (South Africa)[4] and those for 1958 and 1959 were made available by courtesy of the Department.[5] The notifications of diphtheria by province and race were obtained from the same sources and the Department classified the reported cases according to the domicile of the patients in urban or rural districts.[5] The figures from Johannesburg were supplied by courtesy of the City Health Department.[6] For comparative purposes, demographic figures and notifications were also collected from England and Wales,[7] Australia,[8] and Copenhagen,[9] and the annual number of notifications per 100,000 population, hereinafter referred to as the notification rate, was calculated. Where it has been considered sufficient to indicate the size of population groups, the estimates of 1956 have been chosen because of their central position in the observation period.

RESULTS

The estimated populations in the areas under consideration together with the annual notification of diphtheria are shown in Table I. The calculated notification rates, presented graphically in Fig. 1, illustrate clearly the gravity of diphtheria in South Africa, where the observed incidence is 4 times higher than in Australia and 75 times higher than in England and Wales; clinical diphtheria has been eliminated from Copenhagen, and in the United States of America the notification rate fell from 1·9 in 1952 to 1·2 in 1959.[10] Until 1957 the national notification rate in South Africa was remarkably constant, but fell slightly in 1958 and 1959. Diphtheria occurred in all races (Fig. 2). During the period 1953 - 59 the notification rate in Whites decreased by about 60% to 16·69 per 100,000 and the rates in the Asians and Bantu are approaching the same figure. In recent years diphtheria has been observed relatively more frequently among the Coloured population.

Diphtheria has been reported regularly from all 4 provinces (Fig. 3). Despite moderate annual fluctuations in

TABLE I. ANNUAL NOTIFICATIONS OF DIPHTHERIA PER 100,000 POPULATION IN SOUTH AFRICA AND OVERSEAS (POPULATION IN THOUSANDS)

	South Africa			*England and Wales*			*Australia*			*Copenhagen*		
Year	*Population*	*Cases*	*Rate*	*Population*	*Cases*	*Rate*	*Population*	*Cases*	*Rate*	*Population*	*Cases*	*Rate*
1953 ..	13,153	3,228	24·54	44,090	265	0·60	8,918	1,081	12·12	759	0	0
1954 ..	13,427	3,342	24·89	44,274	166	0·37	9,090	704	7·74	755	0	0
1955 ..	13,669	3,382	24·74	44,441	154	0·35	9,313	892	9·58	753	0	0
1956 ..	13,915	3,521	25·30	44,667	51	0·11	9,533	348	3·65	746	0	0
1957 ..	14,167	3,440	24·28	44,907	37	0·08	9,747	237	2·43	739	0	0
1958 ..	14,418	3,304	22·92				9,952	103	1·03	733	0	0
1959 ..	14,673	2,733	18·63									

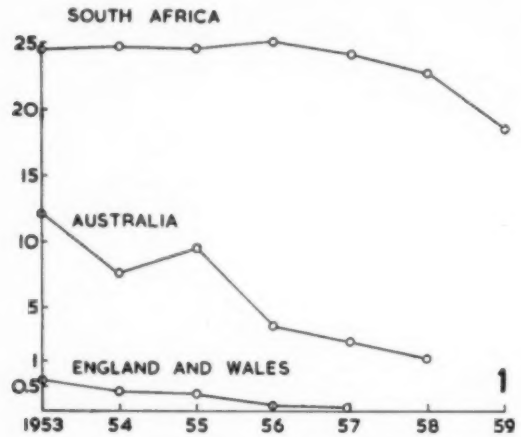

Fig. 1. Annual notifications per 100,000 population in South Africa, Australia, and England and Wales.

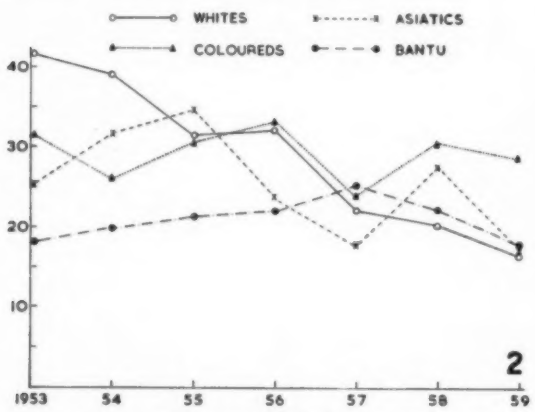

Fig. 2. Racial notification per 100,000 population per annum. (Population in thousands in 1956: White, 2,907; Asian, 431; Coloured, 1,319; Bantu, 9,460.)

notification rates during the observation period, the morbidity appears to have been much the same in each province. This, however, does not exclude the existence of racial foci. Their racial make-up in 1956 is shown in Table II and the notification rates for the groups are plotted in Fig. 4. For comparison a graph giving the mean for the whole country is included in each section. It emerges that the observed incidence of diphtheria in the races varies to some extent with the geographical division. Thus, in Natal and the Transvaal, the White, Asian and Coloured groups appear to have suffered more from diphtheria than in the other provinces. While the notification rates of the Bantu in the Orange Free State were comparatively high, they were below the mean for the Union in all other provinces. More important than these variations, however, is the observation that the disease showed no distinct evidence of racial predilection.

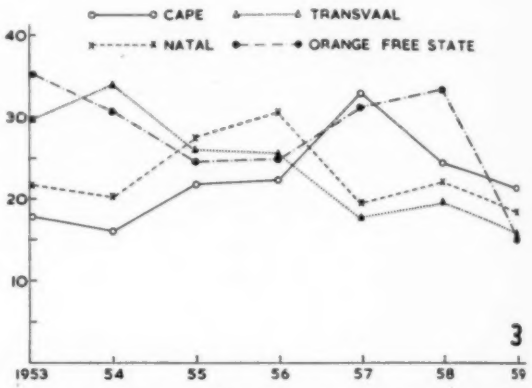

Fig. 3. Annual notifications by province per 100,000 population. (Population in thousands in 1956: Cape, 4,804; Natal, 2,609; Transvaal, 5,346; Orange Free State, 1,156.)

TABLE II. RACIAL COMPOSITION OF THE POPULATION IN THE PROVINCES OF THE UNION OF SOUTH AFRICA, 1956 (POPULATION IN THOUSANDS)

Province	*White*	*Asian*	*Coloured*	*Bantu*
Cape	999	20	1,136	2,649
Natal	311	344	39	1,915
Transvaal	1,344	57	91	3,854
Orange Free State ..	253	—	15	888

Since crowded conditions favour the spread of diphtheria, the disease would be expected to be more common in urban than in rural districts. Estimates of the population so divided are available from 1957 (Table III), but these differ by 2,000 individuals from the numbers given in Table I. While just over half the population lives in rural districts, about 80% of the White and Asian groups, 68% of the Coloured and only 30% of the Bantu group are city dwellers. Calculation of notification rates on this basis shows that the observed diphtheria morbidity is similar in urban and rural districts (Fig. 5). Moreover, the notification rates of the individual

TABLE III. ESTIMATED URBAN AND RURAL POPULATION IN SOUTH AFRICA (POPULATION IN THOUSANDS)

Year	Urban					Rural				
	White	*Asian*	*Coloured*	*Bantu*	*Total*	*White*	*Asian*	*Coloured*	*Bantu*	*Total*
1957	2,395	345	897	2,838	6,475	562	87	423	6,622	7,694
1958	2,439	353	925	2,882	6,599	572	90	435	6,724	7,821
1959	2,485	360	955	2,925	6,725	582	92	450	6,826	7,950

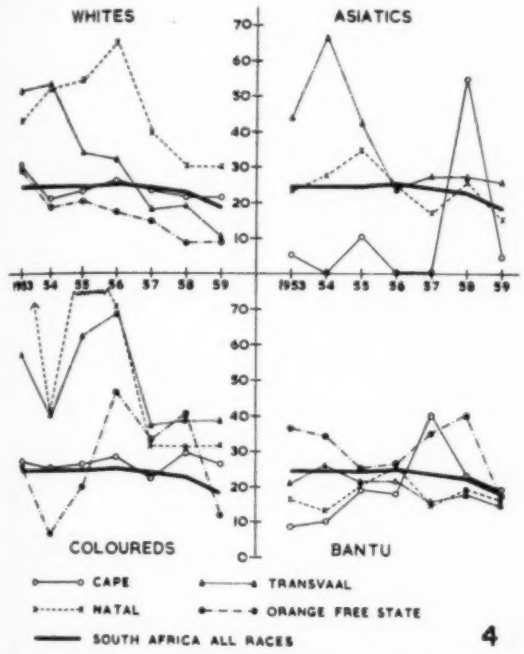

Fig. 4. Racial notification per 100,000 population in the four provinces.

races living under urban and rural conditions respectively (not presented) do not differ substantially from the mean rates shown in Fig. 5.

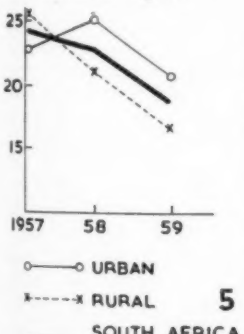

Fig. 5. Urban and rural notification per 100,000 population.

The difficulty of defining urban areas accurately and the incorporation of many small towns in this group,[5] may mask a difference between the occurrence of diphtheria in urban and rural populations. To test this, the notification rates of the country's largest city, Johannesburg, were plotted in Fig. 6. It emerges that, throughout the observation period, diphtheria has been very prevalent in Johannesburg, but the overall rates have declined considerably and now approach the average for the country. Although diphtheria has been reported fairly uniformly from all races in Johannesburg, the Whites have enjoyed the most spectacular fall in notification rates.

Traditionally, the notification rates have been recorded by relating the notifications from a given population to the total number of individuals, although the disease is confined to a certain age group.

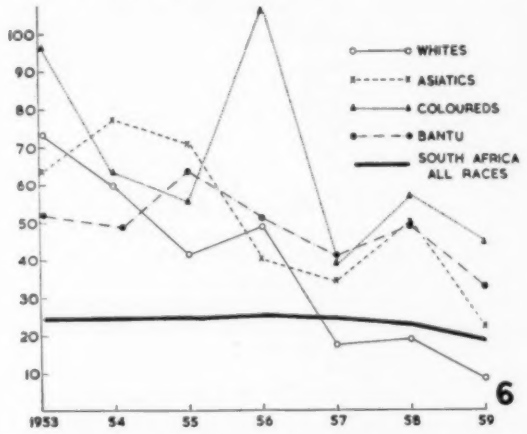

Fig. 6. Racial notification in Johannesburg per 100,000 population. (Population in thousands in 1956: White, 363; Asian, 25; Coloured, 37; Bantu, 539.)

In South Africa practically all cases occur in individuals of less than 20 years of age[3,11] and, since the age distribution of the races, at least in Johannesburg, differs considerably,[20] the crude notification rates may present a distorted picture.[21] This objection has been met in Table IV, which shows the

TABLE IV. RACIAL NOTIFICATION IN JOHANNESBURG PER 10,000 POPULATION UNDER 20 YEARS OF AGE

Year	*White*	*Asian*	*Coloured*	*Bantu*
1953	20·69	11·42	21·14	17·34
1954	16·86	13·86	13·88	16·34
1955	11·67	12·71	12·20	21·22
1956	13·65	7·18	23·59	17·00
1957	4·92	6·21	8·49	13·96
1958	5·36	8·97	12·38	16·63
1959	2·42	3·99	9·76	10·95

notifications related to the population of less than 20 years of age. These fractions were calculated from the annual estimates of the City population[6] on the assumption that the relative age distribution throughout the period was the same as in 1951.[20] It emerges that the disease on the whole has been reported more often from Bantu children, while the observed incidence was very similar in children of other ethnic groups. This conclusion differs from that arrived at by the study of crude notification rates (Fig. 6), in showing that proportionately fewer cases are reported from Coloured than Bantu children.

In Durban, after 1950, the crude notification rate of diphtheria has been highest in the Coloured, followed by

the Bantu, White, and Asian groups.[10] As above, it may be more profitable to relate the notifications to the most susceptible portion of the population. Accordingly, the number of individuals of less than 20 years of age was calculated by relating the age distribution of the 1951 census[22] to the annual estimates of the population.[10,23] On this basis (Table V) it is evident that diphtheria, as in Johannesburg, was observed more often in Bantu children than in Coloured children. This is contrary to observations made from crude notification rates, but is consistent with the fact that 26·02% of the Bantu population is less than 20 years old, while 51·94% of the Coloured population fall in this category. In regard to Whites and Asians, the impressions gained from the crude notification rates[10] are substantiated.

TABLE V. RACIAL NOTIFICATION IN DURBAN PER 10,000 POPULATION UNDER 20 YEARS OF AGE

Year	*White*	*Asian*	*Coloured*	*Bantu*
1950	35·45	7·87	51·06	37·38
1951	14·46	5·59	18·02	42·88
1952	12·10	5·89	8·68	28·04
1953	9·17	5·49	29·11	19·70
1954	17·34	6·31	17·26	27·16
1955	16·26	7·18	35·01	23·40
1956	14·73	7·38	13·00	15·08
1957	8·08	2·72	4·04	7·94
1958	7·92	4·80	4·62	14·48
0 - 19 years old at 1951 census ..	31·00%	57·81%	51·94%	26·02%

DISCUSSION

The reliability of estimations of the morbidity of a disease depends on the accuracy of the census and the reporting of cases. These problems were considered in a recent publication[10] and the view was expressed that in South Africa only minor errors were introduced by census inaccuracies, but there were strong indications of a considerable under-notification of diphtheria, particularly among the non-Whites. Provided that the distribution of types of *C. diphtheriae* was constant among infected individuals, and that the duration of illness before treatment was comparable,[10,11] it was concluded that death rates rather than notification rates might be more reliable standards for the assessment of the incidence of the disease. However, the lack of tabulation of deaths from diphtheria notified from rural non-Whites[4] makes this approach impracticable.

Sporadic cases caused by the gravis type of *C. diphtheriae* have occurred in South Africa,[12-15] but no outbreaks have been reported. Surveying the type distribution of *C. diphtheriae* in Cape Town in 1937, Wright[12] found that 66·1% of the strains from clinical infections belonged to the mitis, and 29·3% to the gravis, type. Of those classified as gravis strains, 75% lacked the characteristic ability to ferment polysaccharides,[16] while this was found in 20% of the mitis strains. Wright's observations[12] have not been confirmed and, in later surveys on the Witwatersrand,[13-15] approximately 90% of the strains were classified as mitis and 5% as gravis. Thus, for calculation purposes, it may be permissible to consider all infections in South Africa to have been caused by the mitis type.

Assuming that the medical facilities for Whites in the country as a whole are similar to those found in Durban, then the case mortality should be expected to be about 3%.[10] The observed mean case mortality in Whites in South Africa for the period 1950 - 57 was 6·8%,[4] which suggests that only 50% of the cases have been reported or that the incidence of diphtheria among Whites is twice as high as revealed by notification rates. Based on an estimated 6% case mortality among non-Whites in Durban,[10] it was calculated that only 33 - 40% of the non-White cases were reported. Since the notification of the disease in the non-White in rural districts is probably less accurate than in Durban, their observed overall case mortality should be about 20% or higher.[10] With the poorer medical facilities prevailing in many rural districts, it may well be that the true case mortality is close to the maximum of 10%,[11] suggesting that the incidence of diphtheria among non-Whites in the whole of South Africa is at least twice as high as that indicated by notification rates. Thus, the deficit in notifications appears to be reasonably uniform throughout the population and, consequently, the notification rates can be assumed to reflect fairly accurately the relative distribution of the disease. It should be pointed out, however, that the picture in Johannesburg and Durban is somewhat distorted by the excess of adults in the Bantu population.

Despite minor racial differences in notification rates in the provinces, distinct ethnic foci of the infection have not been demonstrated. Although Johannesburg proved to be the only locality with a consistently high notification rate, similar conditions have been observed in Durban.[10] In recent years these foci of infection have been greatly reduced, and in 1959 the observed incidence of diphtheria in Johannesburg and Durban did not differ significantly from that of the rest of the country.

Figures on prophylactic immunization relevant to this survey are available only for the White, Asian and Coloured groups in Johannesburg.[17,18] It is interesting to note that the overall immunization rate in Whites born in the period 1950 - 54 was 75·5% and that 64·5% of the children received their primary course before 2 years of age.[18] Although these preventive measures coincide with a reduction in observed incidence (Fig. 6), they were clearly insufficient to eliminate the disease. A similar reduction in notification rates was observed in the Coloured and Asian groups, but in these groups both the overall immunization rates and the proportion immunized before 2 years of age were so low[18] that they could hardly be claimed to have any influence on the morbidity.[17] The Bantu have experienced only a slight decline in notification rate, but though their immunization status is unknown, it is probably safe to assume that it is no better and perhaps worse than that in the Coloured population.

It may be concluded, therefore, that unless the health authorities are able to stimulate the population's interest in immunization, it is probable that the incidence of diphtheria in Johannesburg will remain at a higher level than that prevailing elsewhere. A similar conclusion was drawn from the study in Durban,[10] and there is every indication that it applies to South Africa as a whole.

SUMMARY

Contrary to popular impression, diphtheria has not ceased to be of public concern in South Africa. It appears to be about twice as frequent as indicated by notification rates. Although minor differences in distribution were observed,

it was not possible to detect a distinct focus of the disease in any of the provinces, in the urban or rural districts, or in the ethnic groups. Nor could any district or section of the population claim to have eliminated the disease.

I wish to thank Dr. E. J. H. Foullois, State Health Department, Pretoria, for valuable advice; the City Health Department of Johannesburg for notifications from that city; Mr. N. J. Richardson for technical assistance; and the Director of this Institute, Dr. J. H. S. Gear, for permission to publish this paper.

REFERENCES

1. Grasset, E. (1929): *Diphtheria Prophylaxis. The Use of Anatoxin—Ramon for Immunization against Diphtheria.* Johannesburg: South African Institute for Medical Research.
2. Murray, N. L. (1943): S. Afr. Med. J., **17**, 334.
3. Bokkenheuser, V. and Heymann, C. S. (1954): *Ibid.*, **28**, 685.
4. Department of Health, Union of South Africa (1950 - 57): *Annual Reports.* Pretoria: The Government Printer.
5. Department of Health, Union of South Africa (1960): Personal communication.
6. City Health Department, Johannesburg (1960): Personal communication.
7. Registrar-General (1953 - 57): *Statistical Review of England and Wales. Medical Section.* London: H.M. Stationery Office.
8. Department of Health, Australia (1959): Personal communication from the Director-General, Dr. A. J. Metcalfe.
9. Public Health Department, Copenhagen (1953 - 58): *State of Health in Copenhagen—Annual Report of the Commissioner of Health.*
10. Bokkenheuser, V. and Stephen, A. (1961): S. Afr. Med. J., **35**, 289.
11. Bokkenheuser, V. (1955): *Ibid.*, **29**, 461.
12. Wright, J. (1937): Brit. Med. J., **1**, 259.
13. Murray, J. F. (1942): S. Afr. Med. J., **16**, 247.
14. *Idem* (1943): J. Hyg. (Lond.), **43**, 159.
15. Bokkenheuser, V., von Haebler, T. and Jackson, A. L. (1956): Med. Proc., **2**, 484.
16. Wilson, G. S. and Miles, A. A. (1946): In *Topley and Wilson's Principles of Bacteriology and Immunity*, 3rd. ed., p. 462. London: Arnold.
17. Bokkenheuser, V. (1955): S. Afr. Med. J., **29**, 1249.
18. Bokkenheuser, V. and Smith, A. H. (1960): *Ibid.*, **34**, 928.
19. Moore, H. A. and Larsen, G. I. (1957): Publ. Hlth Rep. (Wash.), **72**, 537.
20. Bokkenheuser, V.: Submitted for publication.
21. Hill, A. B. (1955): *Principles of Medical Statistics*, 6th ed., p. 199. London: The Lancet.
22. Union of South Africa (1951): Population census, vol. 5. Pretoria: Government Printer.
23. City Medical Officer of Health, Durban (1950 - 58): Annual Reports.

HAEMOPERITONEUM ASSOCIATED WITH A SOLITARY DIVERTICULUM OF THE SIGMOID COLON

H. H. LAWSON, F.R.C.S. (ENG.), *formerly Senior Surgical Registrar, St. Helier Hospital, Surrey* *

No report has been found in the literature of a haemoperitoneum occurring during life in association with a diverticulum of the colon, and it was therefore thought that this case should be recorded.

CASE REPORT

History

Mr. J.T., aged 52 years, an outfitter, was admitted to hospital on 31 January 1959. He was complaining of hypogastric pain for 2 days, described as a dull ache. He had vomited once before the pain started, and once afterwards. He had pain in the right shoulder. He was habitually constipated, and since mecamylamine treatment he had taken increasing doses of senna; he had recently noted that his mouth was dry.

Past History

He was found to be hypertensive in 1953, and his blood pressure varied between 250/145 and 160/110 mm.Hg. In 1957 he had a haemoptysis and later a transient dysarthria, from which he made a complete recovery. In 1958 treatment with 'inversine' was started, and he received 7·5 mg. 8-hourly; his blood pressure being stabilized at 130/95 mm.Hg.

Examination

Blood pressure 150/95 mm.Hg. Pulse 96. Temperature 99°F. He had lower abdominal tenderness, maximal in the right iliac fossa, with guarding and rebound tenderness. Bowel sounds were present. On rectal examination he was tender on the right side. The peripheral pulses were present. A provisional diagnosis of acute appendicitis or acute diverticulitis was made.

Operative Findings

An emergency laparotomy was carried out. There were about 500 ml. of unclotted blood in the peritoneal cavity. The source of bleeding was the sigmoid colon where there was a large haematoma on the side of the bowel, not extending into the mesentery. When this was disturbed, fresh bleeding occurred from what appeared to be a longitudinal split in the sero-muscular coat. There was no faecal contamination. Apart from a chronic duodenal ulcer no other abnormality was seen; in particular no other diverticula were noted. A sigmoid colectomy was carried out, and continuity re-established by an end-to-end anastomosis.

Postoperative

He made a good recovery and his bowels worked normally on the fourth day.

Special Investigations

Full blood count and platelet count were normal. A skin and muscle biopsy from the left thigh showed non-specific changes in the vessels, compatible with long-standing hypertension.

* Present address: Baragwanath Hospital, Johannesburg.

Pathology

The specimen consisted of a segment of colon 15 cm. long. On the anti-mesenteric surface of the bowel, between the lateral taenia coli, there was a solitary diverticulum whose base was 3 cm. in diameter; the lining of the diverticulum was intact and it was surrounded by blood clot.

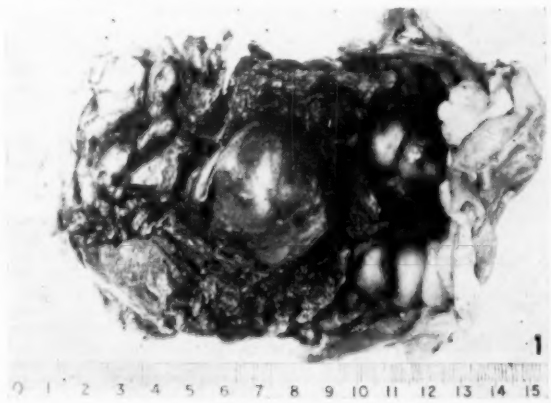

Fig. 1. See text.

Fig. 1 shows the specimen from the serosal aspect—the contracted taenia are clearly seen.

The histological report was as follows: 'Study of 3 sections from the edge of the diverticulum shows both submucosal and subserosal haemorrhage, with separation of mucosa from muscle by blood and fibrin, mixed with polymorphs. At this point the muscle coat, and apparently also the serosa, cease, leaving the diverticular wall consisting of mucosa supported by haemorrhagic submucosa, possibly fused with some serosa. Soon the mucosa is lost, and the wall then consists of necrotic connective tissue, presumably mainly submucosa'.

DISCUSSION

Solitary diverticula of the large bowel have been reported fairly often, by Dorling,[1] Fairbank and Rob,[2] Kirkman,[3] and others, and in these cases the clinical picture was caused by acute inflammation. Although the appendices epiploicae are well supplied with blood vessels, intraperitoneal bleeding

does not occur, presumably because of inflammation causing thrombosis in the peridiverticular vessels.

In the present case the lack of gross inflammatory changes therefore seems to be significant, and it is suggested that the bleeding occurred as a result of the hypertensive changes in the blood vessels themselves, and that the diverticulum was secondary to this change as well, originating in an infarcted area. The histological appearances of small vessels in the colon were identical with those in the muscle biopsy. A similar experience is reported by Woolf and Thompson.[4] In their 3 cases of spontaneous intra-abdominal bleeding, all the patients were hypertensive, with hypertensive changes in the smaller vessels.

It is difficult to decide whether or not mecamylamine played any part, but Addison and Towers[5] have recently reported a case of subserous jejunal haemorrhage during therapy with this drug.

SUMMARY

A case of haemoperitoneum is described, associated with a solitary diverticulum of the sigmoid colon. The patient was a known hypertensive, receiving mecamylamine. The blood vessels showed hypertensive changes, and it is suggested that the diverticulum and haemoperitoneum were secondary to this.

I thank Mr. A. York Mason, under whose care the patient was admitted, for permission to publish this case. I also thank Dr. W. Rogers for his advice and Dr. T. Goodier for the histological report.

REFERENCES

1. Dorling, G. C. (1945): Brit. Med. J., **1**, 513.
2. Fairbank, T. J. and Rob, C. G. (1947): Brit. J. Surg., **35**, 105.
3. Kirkman, N. F. (1950): *Ibid.*, **37**, 365.
4. Woolf, A. L. and Thompson, H. K. (1949): Brit. Med. J., **1**, 572.
5. Addison, N. V. and Towers, J. R. H. (1959): *Ibid.*, **1**, 1167.

IGNAZ PHILIPP SEMMELWEIS

TER HERINNERING AAN SY WERK OOR KRAAMKOORS, WAT 100 JAAR GELEDE VERSKYN HET

H. D. BREDE, DR. MED. PRIV. DOZ. (KÖLN)

Hoof, Departement van Mikrobiologie, Fakulteit van Geneeskunde, Universiteit van Stellenbosch en Karl Bremer-hospitaal, Bellville, Kp.

Ignaz Philipp Semmelweis, ‚die redder van die moeders', verdien 'n eervolle plek in die geneeskundige geskiedenis. In 'n tyd toe die grondslae van die bakteriologie nog nie geskep was nie, was hy die eerste, wat, op grond van die kliniese beeld en van vergelykings met postmortem-ondersoeke, die oorsaak van kraamkoors herken, en gelyktydig ook voorbehoedmaatreëls ontdek het. Sy leerstelling was eenvoudig en vry van spekulasie, sodat 'n mens waardering van sy kollegas sou verwag het. Maar dit was hom vir sy hele lewe ontsê.

Dr. Semmelweis

Semmelweis is op 1 Julie 1818 in Budapest gebore. Sy vader was 'n welgestelde kruidenier en het hom op 19-jarige ouderdom na Wenen gestuur om daar te studeer. Ignaz was eers van plan om hom in die regte te bekwaam, maar na bywoning van 'n paar anatomiese lesings het hy sy planne verander en in die geneeskunde gestudeer. In 1844 het hy gepromoveer en in 1846 'n aanstelling as assistent by die kraamkliniek van die ‚Algemene-hospitaal' in Wenen aanvaar.

Hierdie kliniek is deur Keiser Joseph II in 1784 vir hulpbehoewende vroue gestig. Daar was twee verlosafdelings. In die eerste het geneeskundige studente onderwys ontvang en in die tweede leerlingvroedvrouens. Alhoewel die behandeling in altwee afdelings dieselfde was, was die sterfte in die eerste afdeling (studente) betreklik hoër dan in die tweede (vroedvrouens). Die verskil was so groot dat die gewone mense daarvan geweet het, en hartroerende tonele het dikwels daar afgespeel indien 'n swanger vrou na die eerste afdeling verwys is.

Na 'n ontleding van die statistiese resultate van altwee afdelings, het Semmelweis besef dat daar 'n rede was vir die vrees by verwysing na die eerste afdeling. Maar wat was die verklaring vir hierdie groot verskil? Te midde van sy pogings om die oorsake op te spoor, is sy beskermheer en ou vriend, Kolletschka, die professor in geregtelike geneeskunde, skielik oorlede ten gevolge van 'n bloedvergifting wat hy gedurende 'n outopsie deur die onversigtigheid van 'n student gekry het. Daar was dieselfde orgaanveranderings by die lyk soos Semmelweis dit dikwels by gevalle van kraamkoors gesien het. Derhalwe het Semmelweis tot die gevolgtrekking gekom dat daar ook dieselfde oorsaak moet wees; nl. vergifting deur lyke-materiaal. By Kolletschka het hierdie ‚gif' deur 'n vingerwond in die liggaam binnegedring; by die kraamvrouens word die gif deur ondersoek van geneeshere en studente in die geboorteweë in gebring.

Dit kon dus op hierdie manier verklaar word waarom meer vrouens in die eerste afdeling gesterwe het as in die tweede; die studente het dikwels aan outopsies deelgeneem voordat hulle na die kraamafdeling toe gekom het. Semmelweis het onmiddelik aan 'n kontakinfeksie gedink, en reeds in 1847 in die eerste afdeling 'n ‚hande-ontsmetting' deur middel van chloorwater ingestel, met die gevolg dat die sterfte aan kraamkoors binne 'n paar maande van 12% na 3% gedaal het. Daarna het hy saam met dr. Lautner diereksperimente met konyne uitgevoer. Na die werpsel van jong diere het hulle in die skede van die moederdiere aansteeklike materiaal ingeënt en daarna dieselfde veranderings by die diere gevind as wat hulle by kraamkoors-gevalle gevind het.

Semmelweis se werk het egter geen erkenning gekry nie. Sy hoof, professor Klein, het die hele werk verbied en sy aanstelling nie verleng nie. Verder het hy die bespreking van Semmelweis se ondervindings belet. Onder hierdie omstandighede het Semmelweis geen ander keuse gehad nie as om na sy geboortestad, Budapest, terug te keer. Selfs daar het sy leerstellinge moeilik inslag gevind. Na jare slaag hy egter daarin om die professoraat in die verloskunde te bekom. Met vasberade ywer bring hy uiteindelik die sterfte as gevolg van kraamkoors in sy kliniek tot onder 1%. Vir hom was dit slegs 'n skrale troos, omdat sy leerstellinge selfs nou nog nie algemeen aanvaar is nie. In 'n laaste wanhopige poging publiseer hy in 1861 sy boek: *Die etiologie, begrip en voorbehoeding van kraamkoors*. Hierdie boek is 'n laaste teregwysing vir sy bekrompe kollegas, want kortliks daarna verval hy in 'n endogene psigose en sterf op 13 Augustus 1865 in die sielsieke inrigting, Döbling, buite Wenen, as gevolg van 'n selftoegediende bloedvergifting.

Erkenning het Semmelweis eers gekry nadat Lister die antiseptiese metodes tot sukses gevoer het.

Semmelweis se probleem is ook vandag, ten spyte van antibiotiese middels en aseptiese metodes, nog baie werklik, want hospitaal- en wondinfeksies bly nog steeds op die voorgrond.

BIBLIOGRAFIE

Garrison, F. H. (1917): *History of Medicine*. Philadelphia en Londen: Saunders.
Semmelweis, I. P. (1847): Z. Gesellsch der Ärtze in Wien, **4**, 242.
Idem (1848): *Ibid.*, **5**, 64.
Idem (1861): *Die Ätiologie, der Begriff und die Prophylaxis des Kindbettfiebers*. Pest, Wien en Leipzig: C. A. Hartleben.

UNIVERSITY NEWS : UNIVERSITEITSNUUS

AKADEMIESE JAARDAG, KARL BREMER-HOSPITAAL

Die Vyfde Akademiese Jaardag van die geneeskundige personeel van die Karl Bremer-hospitaal, Bellville, en die Fakulteit van Geneeskunde van die Universiteit van Stellenbosch, wat op 9 en 10 Augustus 1961 in die Burgersentrum, Bellville, Kp. gehou is, het weer baie suksesvol verloop. Bowe en behalwe die personeel van die Karl Bremer-hospitaal en lede van die Universiteit van Stellenbosch, was daar hierdie jaar baie mediese praktisyns uit die omgewing van Bellville en ook uit Kaapstad wat die verrigting bygewoon het. Die referate wat gelewer is, het 'n groot aantal van die belangrikste aspekte van die moderne medisyne behels. Sommige van die bydraes was van algemene aard — die meeste het egter gehandel oor oorspronklike navorsingswerk wat in die verskillende departemente gedoen word.

Twee indrukke het veral uitgestaan: in die eerste instansie was dit duidelik dat die gebruik van Afrikaans, sowel idiomaties as wat betref die terminologie, groot vooruitgang gemaak het. Op hierdie gebied maak die Geneeskundige Skool dus ook 'n besondere bydrae tot die vooruitgang van die mediese vaktaal in Afrikaans. Die tweede indruk is dat daar bewyse is dat daar alreeds goeie navorsingseenhede in die verskillende kliniese departemente ontstaan. In enige opleidingskool is *dit* van groot belang, en onder die leiding van stimulerende en verbeeldingryke kliniese professore behoort eenhede van hierdie aard baie vinnig te ontwikkel.

Soos in die verlede is daar ook 'n aantal films vertoon, en 'n interessante uitstalling deur 'n hele aantal farmaseutiese firmas is georganiseer. Die hospitaal en die Fakulteit van Geneeskunde is groot dank aan hierdie firmas verskuldig vir hulle hulp en ondersteuning.

Die verrigtinge is op Vrydagaand 11 Augustus afgesluit met 'n dinee-dans in die Burgersentrum, Bellville. As gaste by hierdie geleentheid was, o.a., teenwoordig Sy Edele, dr. Albert Hertzog, Minister van Gesondheid; prof. H. B. Thom, Rektor van die Universiteit van Stellenbosch, en mev. Thom; dr. R. Retief, Direkteur van Hospitaaldienste, en mev. Retief; dr. en mev. J. H. Cairnes; dr. A. P. Blignault, Redakteur van die *Suid-Afrikaanse Tydskrif vir Geneeskunde*, en mev. Blignault; mnr. P. H. Meyer, L.P.R. vir Bellville, en mev. Meyer; en mnr. H. M. Pansegrouw, Voorsitter van die Hospitaalraad van die Karl Bremer-hospitaal, en mev. Pansegrouw.

Soos in die verlede is weer 'n bekroning gemaak uit die werk wat voorgedra is. As beoordelaars het opgetree prof. F. du T. van Zijl, Dekaan van die Fakulteit van Geneeskunde van die Universiteit van Stellenbosch; prof. F. Forman van die Universiteit van Kaapstad; prof. H. Brink van die Departement Fisiologie van die Universiteit van Stellenbosch; en dr. A. P. Blignault.

Die F. du Toit van Zijl Medalje is vanjaar toegeken aan dr. M. P. Keet vir haar voordrag oor werk waaraan sy en dr. A. B. Murray, wat tans in Amerika studeer, saam gewerk het. Haar voordrag het gegaan oor ‚Tetanus neonatorum: 'n voorlopige mededeling aangaande behandeling'.

THE BENEVOLENT FUND : DIE LIEFDADIGHEIDSFONDS

The following donations during July 1961 are gratefully acknowledged:
Met dank word die volgende skenkings gedurende die maand Julie 1961 erken:

Votive cards in Memory of : Geloftekaarte ter Nagedagtenis aan:

Dr. Len Jacobson *by* Dr. I. J. Zadikoff and Dr. A. L. Agranat; Mrs. Gwen Jooste *by* P. L. A. Smuts Esq.; Dr. Donald Sichel *by* S.A. Society of Anaesthetists; Dr. A. S. van Coller *by* Drs. B. Crowhurst Archer, Cheetham, and Paterson; Mrs. G. F. Nathan Smith *by* Drs. Lance Knox, Cheetham, Paterson, and C. Weinberg; Mr. J. N. Grobler *by* Dr. and Mrs. R. Theron; Mrs. L. Blumberg *by* Dr. and Mrs. R. L. Kleinman.

Total Received from Votive Cards: / *Totaal Ontvang van Geloftekaarte:* R39.81

Services Rendered to: Dienste Gelewer aan:

Dr. D. J. Sagor *by* Drs. I. Grayce, K. Brauer, W. G. Davis and J. J. Jacobson.

Dr. J. van der Horst *by* Drs. R. G. Sharp and J. A. S. Marr.

Dr. M. Swart *deur* Dr. D. Dodds.

Total Received from Services Rendered: / *Totaal Ontvang van Dienste Gelewer:* R50.05

Donations: Skenkings:

Drs. Harries, Kuschke, Hofmeyr, Dry, McKenzie, De Villiers, Struthers, P. N. Swanepoel, Prof. Kok, Drs. H. C. Brayshaw, N. C. Hopkins, C. L. J. Coulon, C. C. Haupt, E. C. Morley, A. F. Richey, R. R. McKenzie, R. T. Vaughan, J. Smithers, P. de V. Charlton-Perkins, J. P. Cronje, J. Kleinman, R. E. P. Matthews, D. A. C. Kelly, B. J. Kaplan, T. A. V. van Zyl, P. S. Meyrick, V. R. Makgalmele and J. J. W. W. Bodenstein.

College of General Practitioners (Kaap de Goede Hoop) Balance of dinner R7.00
Cape Western Branch M.A.S.A. (collection box) R14.00
Dr. E. Mason Barker R4.00

Total Received from Donations: / *Totaal Ontvang van Skenkings:* R48.62

Grand Total: Groot Totaal: R138.48

BENEVOLENT FUND OF THE MEDICAL ASSOCIATION

CAR COMPETITION

So far, the sale of tickets in the Car Competition, organized for the Benevolent Fund of the Medical Association, has not been as satisfactory as it could be.

Members of the Association are earnestly requested to do their best to sell as many tickets as possible in an attempt to make the best possible use of this great opportunity for the Benevolent Fund to build up its resources. The organizers of this fund-raising effort sincerely cherish the hope that every one of the 100,000 tickets issued will be sold, and that not a single booklet with unused tickets will be returned.

For the purpose of the competition members are reminded that the closing date is 8 September 1961. All tickets must be sent to Triumph Herald Competition, P.O. Box 1909, Durban, to reach that address not later than 8 September. Members are urged to send in as many completed tickets as they can before that date to make the competition as keen as possible.

WORLD LIST OF FUTURE INTERNATIONAL MEETINGS

ALTERATIONS AND ADDITIONS NOTIFIED DURING JULY 1961

French Congress of Urology, Paris, 25 September 1961. Dr. Roger Couvelaire, Président, Congrès Francaise d'Urologie, 44 rue Boileau, Paris 16e, France.

French Surgical Association, 63rd Congress, Paris, 25 - 30 September 1961. Association Francaise de Chirurgie, 12 rue de Seine, Paris 6e, France.

Latin-American Congress on Microbiology, San José, Costa Rica, 12 - 19 December 1961. Dr. J. de Abate, Secretary General, Aparto 1404, San Josè, Costa Rica.

Meeting on Hypertension, Karachi, December 1961. Chief, Voluntary Leader Programs Branch, Office of Cultural Exchange, Dept. of State, Washington, D.C.

International Congress of Comparative Pathology, Paris, 17 - 18 December 1961. Dr. L. Grollet, 63 av. de Villiers, Paris 17e, France.

International College of Surgeons, Amsterdam, 24 - 29 April 1962. Dr. J. Glazenburg, Secretary General, Netherlands Section, A. Perkstraat 57, Hilversum, Netherlands.

PASSING EVENTS : IN DIE VERBYGAAN

Edenvale Hospital. The next Clinical Meeting will be held on Wednesday 30 August 1961, at 8.00 p.m. in the Board Room. The speakers will be Dr. Morris Epstein (Modern first-aid methods in resuscitation — description and demonstration); Dr. S. Brett (Clinical paediatric problems); Dr. V. N. Gordon (Presentation of a case of epileptiform seizures in the aged); and Dr. J. N. Barnard (Pit-falls of general practice).

All those interested are invited to attend the meeting.

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Lede word daaraan herinner dat hulle die Sekretaris van die Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad, sowel as die Registrateur van die Suid-Afrikaanse Geneeskundige en Tandheelkundige Raad, Posbus 205, Pretoria, moet verwittig van enige adresverandering. Versuim hiervan beteken dat die *Tydskrif* nie afgelewer kan word nie. Dit het betrekking op lede wat oorsee gaan sowel as dié wat binne die Unie van adres verander.

* * *

Dr. W. W. Fontanive, M.B., Ch.B. (Pretoria), LL.D. (Jena), M.Med. (Rad.D.) (Pretoria), M.D. (Münster), has commenced practice as a diagnostic radiologist at 335 Robert Koch Medical Building, Pretorius Street, Pretoria. Telephones: Rooms 33645, residence 782007.

Dr. W. W. Fontanive, M.B., Ch.B. (Pretoria), L.L.D. (Jena), M.Med. (Rad.D.) (Pretoria), M.D. (Münster), het op 1 Augustus 1961 begin praktiseer as diagnostiese radioloog te Robert Koch Mediese-gebou 335, Pretoriusstraat, Pretoria. Telefone: Kamers 33645, woning 782007.

* * *

Somerset Hospital. The monthly clinical meeting which was to be held in September has been called off in view of the South African Medical Congress being held in Cape Town.

Dr. M. C. Gordon Grant, M.R.C.O.G., of Johannesburg, left for overseas on 25 August 1961 to attend the Third World Congress of Gynaecology and Obstetrics, which is being held in Vienna from 3 to 9 September 1961. He will also visit Gynaecological Clinics in Rome, Amsterdam, and London, and will return on 9 October. Dr. Gordon Grant will be accompanied by his wife.

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University of Cape Town and Association of Surgeons of South Africa (M.A.S.A.), Joint Lectures. The next lecture in this series will be held on Wednesday 30 August at 5.30 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. Dr. I. N. Marks will speak on 'Augmented histamine tests and their application'. All members of the Medical Association are welcome to attend this lecture.

* * *

Research Forum, University of Cape Town. The next meeting will be held on Thursday 31 August at 4 p.m. in the Tutorial Room of the Pathology Department, Medical School, Observatory, Cape. Dr. I. Macdonald of Guy's Hospital, London, will speak on 'Kwashiorkor — a disorder of high carbohydrate diets?' This will be followed by a Staff Clinical Conference held in the Falconer Lecture Theatre, Groote Schuur Hospital, Observatory, Cape, at 5.15 p.m. All general practitioners and others who may be interested are invited to attend these meetings.

* * *

Dr. Harry Fine, urologist, of Durban, has changed his address to 32 Trust Buildings, Gardiner Street, Durban. His telephone number remains 64063.

Dr. Harry Fine, uroloog, van Durban, het sy adres verander tot Trust-gebou 32, Gardinerstraat, Durban. Sy telefoonnommer bly 64063.

LIEFDADIGHEIDSFONDS VAN DIE MEDIESE VERENIGING

MOTORKOMPETISIE

Die verkoop van kaartjies in die Motorkompetisie wat georganiseer word vir die Liefdadigheidsfonds van die Mediese Vereniging is nie so bevredigend as wat verwag is nie.

Lede van die Vereniging word dringend versoek om hul bes te doen om so baie kaartjies as moontlik te verkoop met die doel om die beste moontlike gebruik te maak van hierdie groot geleentheid vir die Liefdadigheidsfonds om fondse in te samel. Die organiseerders van hierdie geldinsameling koester die sterk verwagting dat elkeen van die 100,000 kaartjies wat uitgereik is, verkoop sal word en dat geen enkele boekie met ongebruikte kaartjies teruggestuur sal word nie.

Wat betref die Kompetisie word Lede daaraan herinner dat die sluitingsdatum 8 September 1961 is. Alle kaartjies moet na die Triumph Herald Kompetisie, Posbus 1909, Durban, gestuur word, en moet hierdie adres nie later nie as 8 September bereik. Dit sal goed wees as lede so baie voltooide kaartjies as hulle maar kan wil instuur om die Kompetisie so sterk moontlik te maak.

BOOK REVIEWS : BOEKBESPREKINGS

BLOOD TRANSFUSION TECHNIQUES

A Laboratory Handbook of Blood Transfusion Techniques. By A. D. Farr, F.I.M.L.T., A.I.S.T. Pp. xi + 135. Illustrated. R1.75 net. London: William Heinemann Medical Books Limited. 1961.

This handbook is intended primarily for the blood transfusion technologist and was written to 'fill a gap in the literature covering the general practice of blood transfusion as it affects the laboratory'. It therefore details various non-serological techniques. The chapters are arranged under the following headings: general apparatus, crystalloid solutions, whole blood, blood products, blood-volume expanders, and transfusion apparatus.

When reading this book one is struck by the vast differences that exist between the established blood transfusion services in South Africa and those in England. This is particularly so when considering the apparatus used, such as donor collection sets, blood bottles and giving sets. In South Africa, with few exceptions, the disposable type of apparatus is used, whereas in England it is still of the re-usable type. Consequently, the author has devoted almost half the book to descriptions of the preparation of anticoagulant solutions, the cleaning, preparation and assembly of blood bottles, giving sets and donor collection sets. From our point of view, therefore, the merit of this book is largely lost. Because of these and several other equally radical differences, it is not possible to recommend this book to the uncritical reader engaged in transfusion technology in South Africa. B.C.E.

LIGHT COAGULATION OF THE RETINA

Light Coagulation. By Gerd Meyer-Schwickerath, M.D. and translated by S. M. Drance, M.B., F.R.C.S. (Eng.). Pp. 114. 55 illustrations. South African selling price: R8.10. South African agents: P. B. Mayer, Box 713, Cape Town and Westdene Products (Pty.) Ltd., Medical Book Dept., P.O. Box 7710, Johannesburg. St. Louis: C. V. Mosby. 1960.

It is to Professor Gerd Meyer-Schwickerath that credit is due for the practical application of light coagulation of the retina to clinical practice.

This translation of his monograph has brought the work into English medical literature. It gives a résumé of the history and development of this method of treatment, culminating in the use of the xenon arc lamp, which is at present used in the commercial model.

The method by which the light-coagulator is used is described in full, followed by a brief description of the histological changes which occur after treatment.

The main groups of retinal holes and tears and retinal and choroidal tumours which are amenable to treatment are described and illustrated, both before and after treatment.

The description of those retinal degenerations likely to lead to detachment, and their prophylactic treatment by this method, is of immense importance.

Finally, a short chapter on the treatment of certain iris lesions and surface tumours at the limbus, completes an authoritative introduction to this rapidly growing subject.

The translation is well presented in a concise style which makes pleasant reading, although there are too many spelling errors. One hopes this will be rectified in subsequent editions.

Ophthalmologists the world over will want to read this monograph, but its appeal should extend also to all practitioners who are interested in retinal surgery. M.L.

MODERN HUMAN GENETICS

Recent Advances in Human Genetics. Ed. by L. S. Penrose, M.A., M.D., F.R.S. Pp. vii + 194. 13 Illustrations. R2.75 net. London: J. & A. Churchill Ltd. 1961.

This latest addition to the 'recent advances' series is a very welcome one. Human genetics has been medical 'news' of recent years, mainly because of the development of 'nuclear sexing' followed by actual chromosome counting. Abnormal haemoglobins have also been subjects of considerable labour, while newer statistical methods have been applied to such considerations as mutation rates. Chromosome mapping is still in its infancy — a rather protracted infancy in the case of the human. These and other subjects are discussed in this most interesting volume, which will be useful to workers in the field by reason of discussions of methods and a good list of references, and will be illuminating to all medical men who wish to go a little way behind diseases and symptoms and signs. W.P.U.J.

BASIC FACETS OF HYPOTHERMIA

Hypothermia and the Effects of Cold. British Medical Bulletin. Vol. 17, No. 1, January 1961. Pp. 78. Illustrated. R2.00. London: The British Council, Medical Department. 1961.

This is a remarkably interesting and authoritative collection of articles dealing with some of the basic and controversial facets of hypothermia. The subjects covered by contributors, mostly British and Canadian, range widely and, since each article has been written by a specialist, the enormous and fascinating field of hypothermia is laid before the reader in a scientific and thought-provoking manner. Considering space limitations, it is surprising to see how detailed the reports are, and there is an extensive bibliography at the end of each to enable the reader to continue exploring a particular aspect of the vast literature available.

The achievements in this field have been considerable and have come about only recently — many of the techniques and facts presented would not have been believed possible in 1955 — but the most dramatic development is that man can tolerate and recover from a body temperature of as low as 10°C.

The importance of a publication such as this one is to present the current status of research to the uninitiated. It is important also because the more original and detailed literature published, the more quickly will the various difficulties and problems be solved with the interchange of information. C.N.B.

ANAESTHETIC ACCIDENTS

Anaesthetic Accidents. By V. Keating, M.B., B.Ch., D.A., F.F.A.R.C.S. Pp. vii + 288. Illustrated. R2.80 net. London: Lloyd-Luke Medical Books Ltd. 1961.

The second edition of Dr. Keating's very readable and informative book on 'Anaesthetic accidents' is a justification of the writer's aim, which is an attempt to review the causes and to indicate the possible prevention and treatment of these distressing incidents.

All aspects of anaesthesia are subjected to scrutiny, and with our increasing knowledge of the pharmacology of the relaxant drugs, the review of the side-actions of relaxants is of particular importance.

An excellent chapter on general anaesthesia and respiratory function — in which the disturbances arising as a result of hypoxia and hypercapnia are discussed — merits the careful attention of all practising anaesthetists.

Emphasis is laid on the importance of the recovery ward in reducing the mortality and morbidity following anaesthesia, and in a short, but very important, chapter on 'Medico-legal considerations', Dr. Keating makes the following significant statement with reference to the death of a patient under an anaesthetic: 'What is required is that the anaesthetist shall exercise due care, having regard to his experience and standing as a specialist'.

The book is well printed, and is brimful of sound judicious advice on numerous problems with which the anaesthetist is confronted from time to time. J.A.

NEUROLOGY

Essentials of Neurology. By John N. Walton, M.D., M.R.C.P. Pp. xvii + 422. R3.00. London: Pitman Medical Publishing Co. 1961.

The medical student of today certainly has a wide choice of text-books in neurology to assist him in acquiring the basic

knowledge which he needs in this field, and to recommend any single one above all others, is a difficult task. A great deal will depend on the individual student, on his type of mind, whether he wants many detailed facts presented to him and which he is prepared to learn off by sheer exercise of memory, or whether he has a tidier, more logical, approach and is prepared to acquire his knowledge from basic information and general principles to which is added the ceaseless acquisition of clinical experience by zealously visiting the wards and repeatedly examining the clinical material available.

This book is by one of the younger neurologists and it is planned in sections, starting with a consideration of cardinal symptoms and signs and including information about the various investigations that are available. The next section is concerned with brief descriptions of specific syndromes and the volume ends with a small section of 40 pages giving an outline of treatment in neurology. The student who decides to rely on this text will not go wrong, and if he uses the references at the end of each chapter he will become quite knowledgeable in neurology. S.B.

ENDOCRINOLOGY

Progress in Endocrinology. Part I. Neuroendocrinology and endocrinology of the thyroid and parathyroid glands. Ed. by K. Fotherby, J. A. Loraine, J. A. Strong and P. Eckstein. Pp. x + 166. Illustrated. R4.50 net. London: Cambridge University Press. 1960.

Progress in Endocrinology. Part II. Biochemistry and biological actions of steroids and other hormones. Ed. by K. Fotherby, J. A. Loraine, J. A. Strong and P. Eckstein. Pp. xi + 167. Illustrated. R4.50 net. London: Cambridge University Press. 1961.

These two volumes are reports of the proceedings of a meeting on endocrinology held at Edinburgh in August 1959.

Part I contains several papers which stress the importance of the hypothalamus and attempt to establish its function more positively. The section on the thyroid gland contains good papers on TSH assay, the treatment of hyperthyroidism, and auto-immunity. A short paper on clinical aspects of hyperparathyroidism is followed by several papers which probe parathyroid function without shedding much new light. The diagnosis of parathyroid dysfunction does not seem to have been facilitated recently.

Part II deals largely with techniques of assay and studies of action of various steroids. G. F. Marrian contributes a concise review of recent advances in oestrogen biochemistry, and Patricia Jacobs of chromosome studies in human intersex. A symposium on the relationship of the endocrine glands to cancer includes papers on hypophysectomy, ovariectomy, adrenalectomy and the use of corticoids, as well as several of a more experimental nature. The conclusions are generally not helpful.

It is difficult to recommend these volumes widely. Certainly they should be stocked in the medical library, but I doubt whether most doctors would find nine rand worth of information in the pair. R.H.

ORTHOPAEDICS

Orthopaedics. By George Perkins, *M.C.*, M.Ch., F.R.C.S. Pp. xiii + 975. 577 illustrations. R12.60 net. London: University of London, the Athlone Press. 1961.

The author is one of the last of that coterie of British orthopaedic surgeons who laid the sound foundations of British orthopaedic practice. The book abounds with the restatement of many well-known principles, some of which had been forgotten or even misquoted in contemporary writings — 'Plus ca change, plus c'est la même chase' (Les Guêpes).

To choose any chapter for specific comment is to be unrealistic, because each one contains so much that is valuable. However, Chapter VI. and in particular Section 40, 'Injuries of the knee joint', will adequately reward the reader, and it may obviate the excision of many undamaged menisci.

Here and there ideas are expressed, as for example on p. 508 where screwing or wiring a patella is advised, which few would accept. These few divergences do not detract from the immense value of the book.

The author's humble reason for writing this book — 'I cannot believe that an orthopaedic surgeon at the end of his active career has not acquired some knowledge worthy of being handed on', is more than adequately fulfilled. It will be of great value to those studying for examinations, both undergraduate and postgraduate, and even to those who are already practising the speciality. It forms a worthy companion to 'Fractures and dislocations', previously published by the same author. A.S.

CORRESPONDENCE : BRIEWERUBRIEK

EXTERNAL CARDIAC MASSAGE

To the Editor: May we comment on the interesting paper 'External cardiac massage' which appeared in the *Journal*?[1] Dr. Milner describes 3 successful cases in which closed-chest massage was used to restart the heart. In 2 of these cases the heart was restarted by a few 'firm thumps' to the lower end of the sternum. Although this manoeuvre was successful, a 'few thumps' does not adequately portray the methods of resuscitation which were described either by Rainer and Bullough[2] or by Kouwenhoven *et al.*[3] Surely the essential action is that the heart should be compressed between the sternum and the vertebral column by the patient's lower limbs or by the doctor's hands in a regularly repeated manner until the heart has resumed its rôle? As in the case of open-chest massage, this act is designed to maintain the circulation as well as to restart the heart, and a few thumps on the sternum as reported by Dr. Milner may give readers a distorted picture of this method. This form of resuscitation has been used successfully by one of us following cardiac arrest in several cases of tetanus neonatorum.

'External cardiac massage' is a resuscitative measure which, if performed in a proper manner, is without question very effective. It can be carried out anywhere and by anyone trained to do it. We feel it necessary to comment on the anaesthetic management of the third case — the cachectic patient of 61 years — for drainage of an empyema thoracis. No premedication is mentioned, but whatever was administered probably had some depressant action. This debilitated patient received thiopentone (200 mg.) and d-tubocurarine chloride (30 mg.), was intubated and then inflated with 50% nitrous oxide in oxygen. Thereafter 1% halothane was administered. Then followed cardiac arrest, external cardiac massage, and an injection of mephenteramine sulphate. Such dangerous excursions into polypharmacy is in our opinion undersirable, unwarranted, unnecessary, and extravagant. Here was a patient suffering from a severe toxaemia with a degree of ventilatory embarrassment enough to indicate rib resection and drainage. This embarrassment was further aggravated by a combination of depressant drugs and by posture. Cardiac arrest was not surprising, but fortunately he recovered.

We feel, with due respect, that this was a case where cardiac arrest was preventable. The use of local or regional block anaesthesia with a few ml. of ½% or 1% xylocaine would have preserved the patient's compensatory protective mechanisms and would not have added to his already embarrassed vital functions.

The drama of cardiac arrest would have been obviated, and further wear and tear on the coronaries of the anaesthetist would have been avoided — and all this at the price of five to ten cents!

B. Jackson
H. Grant-Whyte

S.A. Mutual Building
Gardiner Street
Durban
11 August 1961

1. Milner, L. (1961): S. Afr. Med. J., **35**, 653.
2. Rainer, E. H. and Bullough, J. (1957): Brit. Med. J., **2**, 1024.
3. Kouwenhoven, W. B., Jude, J. R. and Knickerbocker, G. G. (1960): J. Amer. Med. Assoc., **173**, 1064.